10/728766

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     (FILE 'HOME' ENTERED AT 14:18:13 ON 11 APR 2006)

     FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS,
     LIFESCI' ENTERED AT 14:18:37 ON 11 APR 2006
L1         25776 S THIOREDOXIN
L2       7634805 S CLON? OR EXPRESS? OR RECOMBINANT
L3         12217 S L1 AND L2
L4          7209 S HISTIDINE (W) TAG?
L5            83 S L3 AND L4
L6        662353 S DYE?
L7             0 S L5 AND L6
L8            48 S L3 AND L6
L9            37 DUP REM L8 (11 DUPLICATES REMOVED)
L10        50437 S INCLUSION (W) BOD?
L11            1 S L9 AND L10
                 E CHATTERJEE D K/AU
L12          550 S E3-E12
                 E LONGO M/AU
L13          698 S E3
                 E OBERFELDER R/AU
L14           56 S E3-E7
                 E FLYNN E/AU
L15          285 S E3
L16         1578 S L11 OR L12 OR L13 OR L14 OR L15
L17            1 S L9 AND L16

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NEWS 4	JAN 13	IPC 8 searching in IFIPAT, IFIUDB, and IFICDB
NEWS 5	JAN 13	New IPC 8 SEARCH, DISPLAY, and SELECT enhancements added to INPADOC
NEWS 6	JAN 17	Pre-1988 INPI data added to MARPAT
NEWS 7	JAN 17	IPC 8 in the WPI family of databases including WPIFV
NEWS 8	JAN 30	Saved answer limit increased
NEWS 9	FEB 21	STN AnaVist, Version 1.1, lets you share your STN AnaVist visualization results
NEWS 10	FEB 22	The IPC thesaurus added to additional patent databases on STN
NEWS 11	FEB 22	Updates in EPFULL; IPC 8 enhancements added
NEWS 12	FEB 27	New STN AnaVist pricing effective March 1, 2006
NEWS 13	FEB 28	MEDLINE/LMEDLINE reload improves functionality
NEWS 14	FEB 28	TOXCENTER reloaded with enhancements
NEWS 15	FEB 28	REGISTRY/ZREGISTRY enhanced with more experimental spectral property data
NEWS 16	MAR 01	INSPEC reloaded and enhanced
NEWS 17	MAR 03	Updates in PATDPA; addition of IPC 8 data without attributes
NEWS 18	MAR 08	X.25 communication option no longer available after June 2006
NEWS 19	MAR 22	EMBASE is now updated on a daily basis
NEWS 20	APR 03	New IPC 8 fields and IPC thesaurus added to PATDPAFULL
NEWS 21	APR 03	Bibliographic data updates resume; new IPC 8 fields and IPC thesaurus added in PCTFULL
NEWS 22	APR 04	STN AnaVist $500 visualization usage credit offered

NEWS EXPRESS FEBRUARY 15 CURRENT VERSION FOR WINDOWS IS V8.01a,
CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
AND CURRENT DISCOVER FILE IS DATED 19 DECEMBER 2005.
V8.0 AND V8.01 USERS CAN OBTAIN THE UPGRADE TO V8.01a AT
http://download.cas.org/express/v8.0-Discover/

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=> file medline embase biosis biotechds scisearch hcaplus ntis lifesci
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=> s thioredoxin
L1       25776 THIOREDOXIN

=> s clon? or express? or recombinant
L2     7634805 CLON? OR EXPRESS? OR RECOMBINANT

=> s l1 and l2
L3       12217 L1 AND L2

=> s histidine (w) tag?
L4        7209 HISTIDINE (W) TAG?

=> s l3 and l4
L5          83 L3 AND L4

=> s dye?
L6      662353 DYE?

=> s l5 and l6
L7           0 L5 AND L6

=> s l3 and l6
L8          48 L3 AND L6

=> dup rem l8
PROCESSING COMPLETED FOR L8
L9          37 DUP REM L8 (11 DUPLICATES REMOVED)

=> d 1-37 ibib ab
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L9 ANSWER 1 OF 37 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2006137243 EMBASE
TITLE: Database of two-dimensional polyacrylamide gel electrophoresis of proteins labeled with CyDye DIGE Fluor saturation **dye**.
AUTHOR: Fujii K.; Kondo T.; Yokoo H.; Okano T.; Yamada M.; Yamada T.; Iwatsuki K.; Hirohashi S.
CORPORATE SOURCE: Prof. T. Kondo, Cancer Proteomics Project, National Cancer Center Research Institute, 5-1-1 Tsukiji, Chuo-ku, Tokyo 104-0045, Japan. takondo@gan2.res.ncc.go.jp
SOURCE: Proteomics, (2006) Vol. 6, No. 5, pp. 1640-1653. .
Refs: 27
ISSN: 1615-9853 E-ISSN: 1615-9861 CODEN: PROTC7
COUNTRY: Germany
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
029 Clinical Biochemistry
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 5 Apr 2006
Last Updated on STN: 5 Apr 2006

AB CyDye DIGE Fluor saturation **dye** (saturation **dye**, GE Healthcare Amersham Biosciences) enables highly sensitive 2-D PAGE. As the **dye** reacts with all reduced cysteine thiols, 2-D PAGE can be performed with a lower amount of protein, compared with CyDye DIGE Fluor minimal **dye** (GE Healthcare Amersham Biosciences), the sensitivity of which is equivalent to that of silver staining. We constructed a 2-D map of the saturation **dye**-labeled proteins of a liver cancer cell line (HepG2) and identified by MS 92 proteins corresponding to 123 protein spots. Functional classification revealed that the identified proteins had chaperone, protein binding, nucleotide binding, metal ion binding, isomerase activity, and motor activity. The functional distribution and the cysteine contents of the proteins were similar to those in the most comprehensive 2-D database of hepatoma cells (Seow et al., Electrophoresis 2000, 21, 1787-1813), where silver staining was used for protein visualization. Hierarchical clustering on the basis of the quantitative **expression** profiles of the 123 characterzed spots labeled with two charge- and mass-matched saturation **dyes** (Cy3 and Cy5) discriminated between nine hepatocellular carcinoma cell lines and primary cultured hepatocytes from five individuals, suggesting the utility of saturation **dye** and our database for proteomic studies of liver cancer. .COPYRGT. 2006 Wiley-VCH Verlag GmbH & Co. KGaA.

L9 ANSWER 2 OF 37 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2005:1311323 HCAPLUS
DOCUMENT NUMBER: 144:47000
TITLE: Lung endothelial cell associated marker proteins as targets for tissue-specific imaging and therapeutical agents in diagnosis and therapy
INVENTOR(S): Schnitzer, Jan E.; Oh, Phillip
PATENT ASSIGNEE(S): Sidney Kimmel Cancer Center, USA
SOURCE: PCT Int. Appl., 84 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005117977	A2	20051215	WO 2005-US19398	20050602

WO 2005117977 A3 20060202
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
US 2006024231 A1 20060202 US 2005-143114 20050602
PRIORITY APPLN. INFO.: US 2004-576114P P 20040602
AB Methods of delivering an agent in a tissue-specific manner, particularly lung tissue, by targeting a protein **expressed** on the endothelial cell surface, are described. The methods can be used for detecting, imaging and/or treating pathologies, as well as for diagnostics. Specifically claimed are a series of lung endothelial cell associated marker proteins for diagnostic and therapeutical uses, in particular TIE-2, APN, TEM4, TEM6, ICAM-1, nucleolin, P2Z receptor, Trk-A, FLJ10849, HSPA12B, APP, and OX-45.

L9 ANSWER 3 OF 37 MEDLINE on STN
ACCESSION NUMBER: 2005629887 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16159878
TITLE: Endogenous **thioredoxin** is required for redox cycling of anthracyclines and p53-dependent apoptosis in cancer cells.
AUTHOR: Ravi Dashnamoorthy; Muniyappa Harish; Das Kumuda C
CORPORATE SOURCE: Department of Pathology and Arkansas Cancer Research Center, University of Arkansas for Medical Sciences, Little Rock, Arkansas 72205, USA.
CONTRACT NUMBER: 1R01HL071558 (NHLBI)
SOURCE: The Journal of biological chemistry, (2005 Dec 2) Vol. 280, No. 48, pp. 40084-96. Electronic Publication: 2005-09-13. Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200602
ENTRY DATE: Entered STN: 20051129
Last Updated on STN: 20060204
Entered Medline: 20060203
AB Apoptosis is a major mechanism of cancer cell destruction by chemotherapy and radiotherapy. The anthracycline class of antitumor drugs undergoes redox cycling in living cells producing increased amounts of reactive oxygen species and semiquinone radical, both of which can cause DNA damage, and consequently trigger apoptotic death of cancer cells. We show here that MCF-7 cells overexpressing **thioredoxin** (Trx) were more apoptotic in response to daunomycin. Trx overexpression in MCF-7 cells increased the generation of superoxide anion (O2*-) in anthracycline-treated cell extracts. Enhanced generation of O2- in response to daunomycin inTrx-overexpressing MCF-7 cells was inhibited by diphenyleneiodonium chloride, a general NADPH reductase inhibitor, demonstrating that Trx provides reducing equivalents to a bioreductive enzyme for redox cycling of daunomycin. Additionally Trx increased p53-DNA binding and **expression** in response to anthracyclines. MCF-7 cells **expressing** mutant redox-inactive Trx showed decreased superoxide generation, apoptosis, and p53 protein and DNA binding. In addition, down-regulation of endogenous Trx **expression** by small interfering RNA resulted in decreased

expression of caspase-7 and cleaved poly(ADP-ribose) polymerase **expression** in response to daunomycin. These results suggest that endogenous Trx is required for anthracycline-mediated apoptosis of breast cancer cells. Taken together, our data demonstrate a novel pro-oxidant and proapoptotic role of Trx in anthracycline-mediated apoptosis in anthracycline chemotherapy.

L9 ANSWER 4 OF 37 MEDLINE on STN
ACCESSION NUMBER: 2005018546 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15643880
TITLE: Synthesis and characterization of dimaleimide fluorogens designed for specific labeling of proteins.
AUTHOR: Girouard Stephane; Houle Marie-Helene; Grandbois Alain; Keillor Jeffrey W; Michnick Stephen W
CORPORATE SOURCE: Department of Chemistry, Universite de Montreal, C.P. 6128, Succ. Centre-Ville, Montreal, Quebec H3C 3J7 Canada.
SOURCE: Journal of the American Chemical Society, (2005 Jan 19) Vol. 127, No. 2, pp. 559-66.
Journal code: 7503056. ISSN: 0002-7863.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200503
ENTRY DATE: Entered STN: 20050113
Last Updated on STN: 20050304
Entered Medline: 20050303

AB A series of aromatic compounds were prepared bearing two maleimide groups attached directly to the fluorescent cores. The resulting derivatives do not fluoresce until the maleimide groups undergo their typical thiol addition reaction, thus removing their ability to quench fluorescence, as shown by kinetic and spectral characterization studies. In this way, the title compounds serve as fluorogens capable of detection of small thiols or appropriately sized dithiols. **Recombinant** alpha-helical proteins were then designed to bear two cysteine residues capable of regioselective dithiol addition reaction with the dimaleimide fluorogens, thus acting as spatially encoded substrates that form specifically labeled covalent complexes. The efficiency of this in vitro fluorescent protein-labeling reaction demonstrates the feasibility of the development of a method for the fluorescent labeling of specific **recombinant** proteins.

L9 ANSWER 5 OF 37 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:388294 HCAPLUS
DOCUMENT NUMBER: 144:65239
TITLE: Prediction of Nephrotoxicant Action and Identification of Candidate Toxicity-Related Biomarkers
AUTHOR(S): Thukral, Sushil K.; Nordone, Paul J.; Hu, Rong; Sullivan, Leah; Galambos, Eric; Fitzpatrick, Vincent D.; Healy, Laura; Bass, Michael B.; Cosenza, Mary E.; Afshari, Cynthia A.
CORPORATE SOURCE: Amgen Inc., Thousand Oaks, CA, 91320, USA
SOURCE: Toxicologic Pathology (2005), 33(3), 343-355
CODEN: TOPADD; ISSN: 0192-6233
PUBLISHER: Taylor & Francis, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB A vast majority of pharmacol. compds. and their metabolites are excreted via the urine, and within the complex structure of the kidney, the proximal tubules are a main target site of nephrotoxic compds. The authors used the model nephrotoxicants mercuric chloride, 2-bromoethylamine hydrobromide, hexachlorobutadiene, mitomycin, amphotericin, and puromycin to elucidate time- and dose-dependent global gene **expression** changes associated with proximal tubular toxicity.

Male Sprague-Dawley rats were dosed via i.p. injection once daily for mercuric chloride and amphotericin (up to 7 doses), while a single dose was given for all other compds. Animals were exposed to 2 different doses of these compds. and kidney tissues were collected on day 1, 3, and 7 postdosing. Gene **expression** profiles were generated from kidney RNA using 17K rat cDNA dual **dye** microarray and analyzed in conjunction with histopathol. Anal. of gene **expression** profiles showed that the profiles clustered based on similarities in the severity and type of pathol. of individual animals. Further, the **expression** changes were indicative of tubular toxicity showing. hallmarks of tubular degeneration/regeneration and necrosis. Use of gene **expression** data in predicting the type of nephrotoxicity was then tested with a support vector machine (SVM)-based approach. A SVM prediction module was trained using 120 profiles of total profiles divided into four classes based on the severity of pathol. and clustering. Although mitomycin C and amphotericin B treatments did not cause toxicity, their **expression** profiles were included in the SVM prediction module to increase the sample size. Using this classifier, the SVM predicted the type of pathol. of 28 test profiles with 100% selectivity and 82% sensitivity. These data indicate that valid predictions could be made based on gene **expression** changes from a small set of **expression** profiles. A set of potential biomarkers showing a time- and dose-response with respect to the progression of proximal tubular toxicity were identified. These include several transporters (Slc21a2, Slc15, Slc34a2), Kim 1, IGFbp-1, osteopontin, a -fibrinogen, and Gstα.

REFERENCE COUNT: 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 6 OF 37 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2005:1115101 HCAPLUS
DOCUMENT NUMBER: 143:433934
TITLE: Gene **expression** profiling of responses to dimethylarsinic acid in female F344 rat urothelium
AUTHOR(S): Sen, Banalata; Wang, Amy; Hester, Susan D.; Robertson, John L.; Wolf, Douglas C.
CORPORATE SOURCE: Environmental Carcinogenesis Division, National Health and Environmental Effects Laboratory, US Environmental Protection Agency, Research Triangle Park, NC, 27711, USA
SOURCE: Toxicology (2005), 215(3), 214-226
CODEN: TXCYAC; ISSN: 0300-483X
PUBLISHER: Elsevier Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Gene **expression** profiling has been shown to be useful for identifying underlying mechanisms of toxicity, determining patterns of biol. response, and elucidating candidate markers of exposure and response. Inorg. arsenic (iAs) is a human carcinogen and epidemiol. evidence implicates it in the development of urinary bladder cancer. Dimethylarsinic acid (DMA), the major excreted metabolite of iAs in humans, is a known rat bladder carcinogen. To examine the changes associated with DMA exposure, microarray anal. of the urothelium was performed in female F344 rats exposed to non-toxic and toxic doses of DMA in their drinking water for 28 days. A novel method for isolating predominantly urothelial cells was developed. Gene **expression** profiling of the urothelium using a custom 2-**dye** spotted array revealed that DMA treatment modulated the **expression** of transcripts of genes that regulate apoptosis, cell cycle regulation and the oxidative stress response. **Expression** of genes mapping to pathways involved in cancer control processes were also altered after DMA exposure. Morphol. data suggested a dose dependent increase in cellular toxicity. Significant changes in differential gene **expression** were present after all treatments event at doses where standard toxicol. responses were not

detectable. The greatest perturbation in gene **expression** was present in rats after treatment with 40 ppm DMA. Doses which produced no histol. or ultrastructural evidence of toxicity (non-toxic) could be differentiated from toxic doses based on the **expression** of a subset of genes, which control cell signaling and the stress response. These reported changes in gene **expression** show similarities between the mechanisms of action of DMA in vivo and those previously described for iAs in vitro. These data illustrate the utility of transcriptional profiling and its potential in predicting key mechanistic pathways involved in toxicity and as a time efficient tool to inform the mode of action anal. in risk assessment.

REFERENCE COUNT: 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 7 OF 37 BIOTECHDS COPYRIGHT 2006 THE THOMSON CORP. on STN

ACCESSION NUMBER: 2004-13947 BIOTECHDS

TITLE: Novel polypeptide antagonist to peripheral-type benzodiazepine receptor, useful for treating disease or condition such as breast cancer, multiple sclerosis, cirrhosis, ischemia and Alzheimer's disease; antagonist against receptor for use in disease therapy and gene therapy

AUTHOR: PAPADOPOULOS V; GAZOULI M

PATENT ASSIGNEE: UNIV GEORGETOWN

PATENT INFO: WO 2004031722 15 Apr 2004

APPLICATION INFO: WO 2003-US29822 23 Sep 2003

PRIORITY INFO: US 2002-414635 1 Oct 2002; US 2002-414635 1 Oct 2002

DOCUMENT TYPE: Patent

LANGUAGE: English

OTHER SOURCE: WPI: 2004-348000 [32]

AB DERWENT ABSTRACT:

NOVELTY - A polypeptide antagonist (I) to the peripheral-type benzodiazepine receptor (PBR), is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for: (1) identifying (M1) an antagonist to the PBR, involving: (a) obtaining a library comprising random polypeptides; (b) screening the library to identify polypeptides that bind to PBR; and (c) testing the identified polypeptides for the ability to prevent binding of a recognized PBR ligand, where the identified polypeptide that prevents binding of a PBR ligand is an antagonist of PBR; (2) an antagonist (II) of the PBR identified by (M1); (3) screening (M2) for compounds which are antagonists to the PBR receptor, involving: (a) attaching PBR or its biologically-active polypeptide fragment to a substrate; (b) exposing the PBR or its biologically-active polypeptide fragment to one or more compounds; (c) determining whether the compound is bound to the PBR or its biologically-active polypeptide fragment; and (d) if the compound is bound, further determining whether the compound modulates the interaction between the PBR ligand and the PBR; (4) a compound identified by (M2); (5) a composition (III) for treating a PBR-related disease or condition, comprising a compound which acts as an antagonist to the PBR; (6) preparing (III), involves combining an antagonist to the PBR and a carrier; (7) a kit (IV) comprising an antagonist to the PBR; (8) a compound identified using (II); (9) an isolated polypeptide (V) comprising a motif (MF) Ser-Thr-XXXX-Pro, where the polypeptide modulates PBR and X represents any amino acid; (10) an antibody (VI) or its biologically-active fragment which recognizes and binds to PBR antagonist amino acid sequence which contains MF; (11) a nucleic acid (VII) encoding (I); (12) a vector comprising (VII); and (13) an antagonist of the PBR which is not a polypeptide, where the agonist is a peptide mimetic designed to correspond to a polypeptide antagonist of PBR.

WIDER DISCLOSURE - Detecting the ability of a test sample to affect the binding interaction of a first polypeptide and a second polypeptide of a polypeptide binding pair, where the first polypeptide is a PBR antagonist polypeptide and the second polypeptide is PBR is also

disclosed.

BIOTECHNOLOGY - Preferred Antagonist: (I) blocks agonists of the PBR by competitive binding at the ligand binding site of the PBR, where the polypeptide contains MF, and X represents any amino acid. The polypeptide comprises an amino acid sequence (S1) Ser-Thr-Pro-His-Ser-Thr-Pro, which is linked to the TAT sequence derived from HIV. Preferred Kit: In (IV), the antagonists comprises a polypeptide which contains MF. The polypeptide comprises (S1). Preferred Antibody: In (VI), the amino acid sequence comprises (S1). Preferred Polypeptide: (V) comprises (S1), and further comprises TAT sequence of HIV. Preferred Nucleic Acid: In (VII), the polypeptide antagonist is an aptamer, comprising a nucleic acid encoding a scaffold protein in-frame with the polypeptide aptamer. The scaffold protein is **thioredoxin** A.

ACTIVITY - Cytostatic; Neuroprotective; Tranquilizer; Antidepressant; Nootropic; Hepatotropic; Vasotropic; Antiparkinsonian; Vulnerary.

MECHANISM OF ACTION - Antagonist of PBR; Blocks agonist of PBR by competitive binding at the ligand binding site of the PBR; Regulator of PBR activity (claimed). In vitro analysis of PBR antagonist in inhibiting steroid biosynthesis, was carried out as follows: MA-10 Leydig cells were incubated in the presence of increasing concentrations of TAT-Ser-Thr-Pro-His-Ser-Thr-Pro for 30 minutes. Cells were then exposed to 1 microM of Ro5-4864 (4'-chlorodiazepam). After 4 hours, steroid secreted into the media, was measured by radioimmunoassay. The result indicated inhibition of steroid formation by the sequence with an ED50 of 5 microM. Thus, the sequence was found to act as a competitive PBR antagonist.

USE - (I) is useful for treating a PBR-related disease or condition in a subject such as canine, feline, ovine, equine, porcine, caprine, camelid, avian, bovine, amphibian, fish, murine or primate organism such as human, which involves administering (I) to the subject, where the PBR-related disease or condition is chosen from breast cancer, colon cancer, prostate cancer, multiple sclerosis, alcohol withdrawal, affective disorders, anxiety disorders, seizures, brain tumors, Alzheimer's disease, stress, acute myeloid leukemia, liver cancer, cirrhosis, traumatic brain injury, ischemia, reactions associated with chemical toxins, and their combinations. (I) is useful for regulating a PBR activity in vivo, which involves administering (I), where (I) modulates PBR by binding with the ligand binding site of PBR. (II) is useful for identifying one or more agents that modulate PBR ligand binding, which involves binding a PBR or a ligand binding domain of PBR to a solid support, adding (II) to the PBR or its fragment which binds to PBR in the presence of the agent, measuring the amount of the ligand binding domain of PBR bound to (II) on the support in the presence of the agent, and identifying an agent that reduces the amount of the ligand binding domain of PBR binding to (II) on the support, where the solid substrate is a microarray. (III) is useful for treating the above-mentioned diseases (claimed). (I) is useful for treating disease or condition such as Parkinson's disease and ovarian cancer.

ADMINISTRATION - (I) is administered by inhalation, subcutaneous, intramuscular, intravenous injection, or by oral route. No specific dosage details are given.

EXAMPLE - Purified human chorionic gonadotrophin (hCG) was obtained. MA-10 cells were grown in modified Waymouth's MB752/1 medium containing 15% horse serum. Mitochondria were isolated. A polypeptide library comprising 1.28 x 10 to the power of 9 possible 7-mer sequences, were obtained. As protein target, MA-10 isolated mitochondria were used. In brief, a microtiter well was coated overnight with 150 microl of 100 microg/ml MA-10 mitochondria in 1X phosphate buffered saline (PBS), using 0.1 M sodium bicarbonate (pH of 8.6) and blocked with a blocking solution containing 0.1 M sodium bicarbonate (pH of 8.6), 5 mg/ml bovine serum albumin (BSA) and 0.02% sodium nitrite. Peripheral-type benzodiazepine receptor (PBR) function on coated mitochondria was examined using radioligand binding assays. About 2 x 10 transducing units of phage from

the library were combined and incubated for 1 hour with the coated well at room temperature in 100 microl TBST buffer (50 mM Tris-hydrochloric acid (pH of 7.6), 150 mM sodium chloride and 0.1% (v/v) Tween-20). Wells were washed 10 times with TBST and eluted with 0.1 M of 1-(2-chlorophenyl)-N-methyl-N- (1-methyl-propyl)-3-isoquinoline carboxamide (PK 11195). DNA from individual phage **clones** was sequenced after three rounds of selection. Single-stranded DNA from individual phage **clones** was purified. Nucleotide sequences were analyzed on a DNA sequencer using the sequencing primer having sequence of 5'-HO-CCCTCATAGTTAGCGTAACG-3'. Radioligand binding assays were performed. MA-10 mitochondria were suspended in 1X PBS at a final concentration of 10 microg protein/100 microl. Specific radioligand binding (2.5 nM) to MA-10 mitochondria was measured in the presence or absence of increasing concentrations (10 to the power of -6 M to 10 to the power of -3 M) of competing PBR peptides. In all cases after 120 minutes of incubation at 4 degrees C, the incubation reaction was stopped by filtration through filters. Bound radioactivity was determined by liquid scintillation spectrometry. Twenty-mer TAT-Ser-Thr-Pro-His-Ser-Thr-Pro and TAT-Ser-Thr-His-Glu-Glu-Thr-Pro peptides were synthesized that contained an NH2-terminal 11-mer TAT protein transduction domain, followed by two glycine residues. Transduction experiments were performed. To determine the efficiency of TAT polypeptide incorporation into the cells, MA-10 cells were cultured overnight on 8-chambered Super Cell Culture Slides at a concentration of approximately 25000 cells/chamber. Media were replaced 24 hours later with fresh media and cells were treated with various concentrations of Oregon Green 488-labeled peptides for various time periods. After the incubation period, cells were washed with PBS and examined by fluorescent microscopy. For steroid synthesis experiments, MA-10 cells were plated into 96-well plate at the density of 2.5 x 10 to the power of 4 cells/well. About 24 hours later, media was replaced with fresh media and cells were treated with the indicated concentrations of peptides for 30 minutes. Cells were then stimulated with 50 ng/ml hCG or 20 microM of the hydrosoluble analog of cholesterol, 22 R-hydrocholesterol in serum-free media for 2 hours. At the end of the incubation, culture media were collected and tested for progesterone production by radioimmunoassay using anti-progesterone antisera. Progesterone production was normalized against the amount of protein in each well. Proteins were quantified using **dye**-binding assay with bovine serum albumin as standard.
In biopanning experiments, PBR interacting phage peptides were eluted using PK 11195 or Ro5-4864 (4'-chlorodiazepam). After three rounds of panning, 20 individual polypeptide **clones** from each eluate were selected, amplified and sequenced. The polypeptide sequences were found to comprise a motif Ser-Thr-XXXX-Pro. (31 pages)

L9 ANSWER 8 OF 37 MEDLINE on STN
ACCESSION NUMBER: 2004513579 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15378754
TITLE: Action and reaction: Chlamydophila pneumoniae proteome alteration in a persistent infection induced by iron deficiency.
AUTHOR: Wehrl Wolfgang; Meyer Thomas F; Jungblut Peter R; Muller Eva-Christina; Szczepek Agnes J
CORPORATE SOURCE: Department of Molecular Biology, Max-Planck Institute for Infection Biology, Berlin, Germany.
SOURCE: Proteomics, (2004 Oct) Vol. 4, No. 10, pp. 2969-81. Journal code: 101092707. ISSN: 1615-9853.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200503
ENTRY DATE: Entered STN: 20041015
Last Updated on STN: 20050303

Entered Medline: 20050302

AB Chlamydophila pneumoniae is an obligate intracellular pathogen implicated in a variety of acute and chronic diseases. Long-term infections are associated with a persistent life stage, in which bacteria can stay for years. They are less accessible to antibiotic treatment but still prone to sustain an inflammatory response. Different in vitro models have been established to mimic and characterize chlamydial persistency. For C. pneumoniae and Chlamydia trachomatis, altered metabolic activities and changed antigenic profiles compared to acute infections have been reported. Most studies including transcriptome and proteome analyses describe persistency induced by IFNgamma treatment. Here, we use iron depletion of the infected cell culture that also leads into persistent infection. We describe differently regulated proteins found by subtractive proteome analysis comparing two early stages of infection with and without addition of the iron chelator deferoxamine-mesylate. While only one bacterial protein was up-regulated during iron deficiency up to 24 h post infection (p.i.), 11 were found to be up-regulated and eight to be down-regulated from 24-48 h p.i. Two down-regulated proteins could be identified by peptide mass fingerprinting as **thioredoxin** reductase and chromosome partitioning protein (ParB). The latter is involved in chromosome segregation. Thus, using a comparative approach we identified on a proteome level down-regulation of ParB in persistent chlamydial forms, which is in agreement with previous results describing changes in cell division and atypical altered morphology of persistent Chlamydiae.

L9 ANSWER 9 OF 37 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2004:446076 SCISEARCH
THE GENUINE ARTICLE: 818YM
TITLE: Oxidant-injured airway epithelial cells upregulate **thioredoxin** but do not produce interleukin-8
AUTHOR: Oslund K L; Miller L A; Usachenko J L; Tyler N K; Wu R; Hyde D M (Reprint)
CORPORATE SOURCE: Univ Calif Davis, Sch Vet Med, Ctr Comparat Resp Biol & Med, 1 Shields Ave, Davis, CA 95616 USA (Reprint); Univ Calif Davis, Sch Vet Med, Ctr Comparat Resp Biol & Med, Davis, CA 95616 USA; Univ Calif Davis, Sch Vet Med, Dept Anat Physiol & Cell Biol, Davis, CA 95616 USA
COUNTRY OF AUTHOR: USA
SOURCE: AMERICAN JOURNAL OF RESPIRATORY CELL AND MOLECULAR BIOLOGY , (MAY 2004) Vol. 30, No. 5, pp. 597-604. ISSN: 1044-1549.
PUBLISHER: AMER THORACIC SOC, 1740 BROADWAY, NEW YORK, NY 10019-4374 USA.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 29
ENTRY DATE: Entered STN: 4 Jun 2004
Last Updated on STN: 4 Jun 2004

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB We tested the hypothesis that oxidant-injured cells upregulate **thioredoxin**, whereas oxidant-stressed, but not injured, cells upregulate interleukin (IL)-8 after injury. We exposed primary human tracheobronchial epithelial cells and transformed human bronchial epithelial cells (BEAS-2B S.6) to 0, 200, 400, or 600 muM H2O2 for 1 h followed by an additional 7 h of incubation. Subsequently, the cells were double-labeled with markers of injury (either Ethidium Homodimer-1 for cellular injury or MitoTracker **dye** for functional mitochondria) or oxidant stress (5-[and 6]-chloromethyl-2',7'-dicholorodihydrofluorescein diacetate) and antibodies specific for the chemoattractants IL-8 or **thioredoxin**. We found significant inverse relationships between numbers and stained chemoattractant volumes of IL-8 and **thioredoxin**-positive cells with increasing H2O2

dose. Cells with mitochondrial injury produced **thioredoxin** but not IL-8, and oxidant-stressed cells were more likely to produce **thioredoxin** than IL-8. Isolated human neutrophils were more likely to colocalize with **thioredoxin**-positive BEAS-2B S.6 cells than **thioredoxin**-negative cells. The H2O2 injury did not induce significant apoptosis in the BEAS-2B S.6 cells as measured by caspase 3 activation. We conclude that oxidant-injured and stressed airway epithelial cells upregulate **thioredoxin**, but produce little IL-8, which may be important in airway epithelial cell-mediated multistep navigation of neutrophils to sites of oxidant injury.

L9 ANSWER 10 OF 37 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2005:524860 BIOSIS
DOCUMENT NUMBER: PREV200510314823
TITLE: **Thioredoxin**-interacting protein controls endothelial cell apoptosis and oxidative stress in hyperglycemia.
AUTHOR(S): Schulze, P. Christian [Reprint Author]; Yoshioka, Jun; Lee, Richard T.
CORPORATE SOURCE: Harvard Univ, Sch Med, Brigham and Womens Hosp, Boston, MA USA
SOURCE: Circulation, (OCT 26 2004) Vol. 110, No. 17, Suppl. S, pp. 260.
Meeting Info.: 77th Scientific Meeting of the American-Heart-Association. New Orleans, LA, USA. November 07 -10, 2004. Amer Heart Assoc.
CODEN: CIRCAZ. ISSN: 0009-7322.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 1 Dec 2005
Last Updated on STN: 1 Dec 2005

AB Increased oxidative stress plays a central role in the progression of diabetic vascular disease. A major thiol-reducing cellular mechanism is the antioxidative **thioredoxin** system which is regulated by its inhibitor **thioredoxin**-interacting protein (Txnip). In addition to its antioxidative function, **thioredoxin** controls apoptotic cellular pathways by binding and inactivating apoptosis-signaling kinase-1 (ASK-1). We investigated the role of Txnip/**thioredoxin** in hyperglycemia-induced oxidative stress and apoptosis. Incubation of bovine aortic endothelial cells (BAEC) with D-glucose increased reactive oxygen species (ROS) measured by flow cytometry using the redox-sensitive **dye** DCFDA (186 +/- 10% vs. controls; p<0.001). In addition, **thioredoxin** activity assessed by the insulin-reduction-assay showed a glucose-dependent decrease (-36.8 +/- 23.4%, p<0.05 vs. controls). Hyperglycemia induced robust gene **expression** of Txnip (>10-fold after 4h at 22.4 mM glucose; p<0.05 vs. controls) and increased protein levels of Txnip by 640% (p<0.05 vs. controls). In contrast, mRNA and protein levels of **thioredoxin** were not altered by glucose. Since Txnip competes with the pro-apoptotic protein ASK-1 for binding to **thioredoxin**, we investigated the effects of Txnip on endothelial apoptosis Hyperglycemia increased apoptosis in endothelial cells (sub-G1 phase: 3.91 +/- 2.55 vs. 1.48 +/- 0.3 % in controls; p<0.05). Adenoviral overexpression of Txnip increased apoptosis by 4.58 +/- 0.6% vs. 3.01 +/- 0.7% in controls (p<0.001). Increased endothelial apoptosis by Txnip overexpression was confirmed by annexin V staining (p<0.05 vs. controls). Immunoprecipitation revealed reduced ASK-1/**thioredoxin** interaction in cells overexpressing Txnip indicating reduced binding of ASK-1 to **thioredoxin** in the presence of increased cellular levels of Txnip. Intriguingly, gene silencing of Txnip resulted in a reduced endothelial cell apoptosis in hyperglycemia (-49 +/- 12%; p<0.001 vs. scrambled RNAi). Thus, glucose-induced **expression** of Txnip inhibits the antioxidative

thioredoxin system, promoting oxidative stress and apoptosis in hyperglycemia. Our findings demonstrate a new potential mechanism of hyperglycemia-induced vascular injury.

L9 ANSWER 11 OF 37 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2005:480423 BIOSIS
DOCUMENT NUMBER: PREV200510267008
TITLE: An experimental study of gene **expressing** profile in human myeloma cell line RPM18226 induced by thalidomide.
AUTHOR(S): Zhang, Mei [Reprint Author]; Liu, Ting; Yang, Bingjing; He, Pengcheng; Wang, Mengchang
CORPORATE SOURCE: Xian Jiaotong Univ, Hosp 1, Xian 710049, Shaanxi, Peoples R China
SOURCE: Blood, (NOV 16 2004) Vol. 104, No. 11, Part 2, pp. 173B. Meeting Info.: 46th Annual Meeting of the American-Society-of-Hematology. San Diego, CA, USA. December 04 -07, 2004. Amer Soc Hematol. CODEN: BLOOAW. ISSN: 0006-4971.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
OTHER SOURCE: GenBank-NM015685; GenBank-AK025983; GenBank-NM000994; GenBank-NM001551; GenBank-NM001033; GenBank-NM001976; GenBank-NM003330; GenBank-NM005167; GenBank-NM005053; GenBank-NM003564; GenBank-NM017432; GenBank-NM001465; GenBank-NM002388; GenBank-BC008861; GenBank-NM001640; GenBank-NM020040; GenBank-NM004341; GenBank-NM003335; GenBank-NM004184
ENTRY DATE: Entered STN: 16 Nov 2005
Last Updated on STN: 16 Nov 2005

AB Multiple myeloma (MM) is one of malignant plasmacyte neoplasm in hematopietic system. Although nearly 70% patients of myeloma response to chemotherapy, but repeated therapies will induce drug resistance soon and lead to refractory or relapsed myelomas. In recent years, thalidomide is used to treat relapsed and refractory myeloma with satisfied effects and overall therapeutic rate with thalidomide is about 60%. Furthermore, the adverse effects of thalidomide is slight without myelosuppression, hepatotoxicity and renal toxicity. So thalidomide is likely to be a prospective antitumor agent. However, the mechanism of antitumor activity of the agent is still not clear. DNA microarray technology hasprovided us a very useful method to detect simultaneously the **expression** pattern of thousands of genes for investigating the molecular antitumor mechanism of thalidomide.To investigate the genes **expression** profiles of multiple myeloma cell line RPMI8226 treated with thalidomide, cDNA microarray were used to detect thousands of gene **expression** in a chip. Two cDNA probes were prepared through reverse transcription from mRNA of RPMI8226 cells with or without thalidomide treatment.The probes were labled with Cy3 and Cy5 fluorescence **dyes** individually,hybridized with cDNA microarray representing 1152 different human genes. Fluorescent intensity were scanned and screened by means of differential analysis between two gene **expression** profiles. After 72 hrs' co-culture of RPMI8226 cells and thalidomide in 100 mu mol/L concentration, the **expression** of 18 genes were up-regulated and 4 genes were down-regulated. The up-regulated genes(GeneBank Accession) included: 1) protein synthesis-related genes: NM_004184(WARS), NM_003335(UBEIL); 2) immune-related protein:NM_001465(FYB), NM_004341 (CAD), NM_002388(MCM3); 3) matabolism related genes: BC008861, NM_001640(APEH), NM_020040(TUBB4Q), NM_001033(RRM1), NM_001976(ENO3), NM_003330 (TXNRDI); 4) cell signals and transducing proteins: NM_005167(ARHC), NM_001465(FYB); 5) other genes:NM_ 017432(PTOV1), NM_003564 (TAGLN2), NM_005053(RAD23A), NM_ 001033(RRM1), AK025983, NM_015685 (**CLONE24904**),NM_033158 (HYAL2). The down-regulated genes includes: 1) protein synthesis-related genes: NM_000994(RPL32); 2).

immune-related proteins: NM_001551 (IGBP1); 3) other genes: NM_002983(SCYA3),NM_002421(MMP1).These genes were involved in preotein synthesis and degradation, cell signal transduction, cytoskeletal movement immune cell matabolism and regulation of anti-oncogene. WARS gene encoding tryptophanyl-tRNA synthetase was up-regulated by thalidomide, while MMP1 gene encoding matrix metalloprotein 1 was down-regulated. They may be related to the inhibition of angiogenesis caused by thalidomide. SCYA3 gene encoding macrophage inflammatory protein-1 alpha was down-regulated by thalidomide, as well as IGBPI gene which encoding immunoglobulin binding protein I. They may play a role in the inhibition of cell proliferation caused by thalidomide. TUBB4Q gene encoding tubulin beta 4, UBEIL gene encodingubiquitin- activating enzyme El-like protein and TXNRDI gene encoding **thioredoxin** reductase I were up-regulated by thalidomide. They may involve in apoptosis of RPM18226 cells induced by thalidomide. FYB gene encoding Fyn-binding protein was up regulated by thalidomide. The elevated **expression** of this gene mayplay a role in the killing of RPM18226 cells by thalidomide.

L9 ANSWER 12 OF 37 EMBASE

ACCESSION NUMBER: 2004449221 EMBASE
TITLE: Quantitative proteome analysis in benign thyroid nodular disease using the fluorescent ruthenium II tris(bathophenanthroline disulfonate) stain.
AUTHOR: Berger K.; Wissmann D.; Ihling C.; Kalkhof S.; Beck-Sickinger A.; Sinz A.; Paschke R.; Fuhrer D.
CORPORATE SOURCE: fued@medizin.uni-leipzig.de
SOURCE: Molecular and Cellular Endocrinology, (30 Nov 2004) Vol. 227, No. 1-2, pp. 21-30. .
Refs: 14
ISSN: 0303-7207 CODEN: MCEND6
PUBLISHER IDENT.: S 0303-7207(04)00309-0
COUNTRY: Ireland
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
022 Human Genetics
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 4 Nov 2004
Last Updated on STN: 4 Nov 2004

AB Thyroid tumorigenesis involves qualitative and quantitative changes in protein **expression**, which can be comprehensively studied by proteome analysis. However, one of the technical bottlenecks of proteomics remains a reliable, sensitive and inexpensive method for quantification of differentially **expressed** proteins. This is due to the limited linear range of most available protein stains, i.e. silver and Coomassie blue, and high costs of commercially available fluorescent stains. In this paper we describe our experience with a lab-made ruthenium based fluorescent stain (ruthenium II tris(bathophenanthroline disulfonate) (RuBPs)) to perform proteome analysis of nodular thyroid disease. We first compared the properties of RuBPs with two highly sensitive protein stains: (1) silver staining and (2) the commercially available fluorescent **dye** Sypro Ruby. We show that in addition to its highly sensitive staining capabilities similar to Sypro Ruby and silver (2 ng), RuBPs offers several advantages such as a broad dynamic range (similar to Sypro Ruby and 500 times broader than the dynamic range of silver stain), low costs ((euro) 0.03 per gel) and excellent compatibility with mass spectrometry. We then applied the inexpensive RuBPs stain to 2D gels (pH 4-7) of four benign thyroid nodules and normal thyroid tissue. We were able to detect .apprx.1800 protein spots/gel in our thyroid samples. Quantitative changes in protein **expression** levels of at least 20-42 proteins were noted in the benign nodules compared with the normal thyroid tissue of the same

patient. Differentially **expressed** spots were further characterised by nano-LC-FTICR and MALDI-TOF mass spectrometry. In summary we demonstrate, that the novel fluorescent ruthenium II tris(bathophenanthroline disulfonate) stain is a highly sensitive, reliable and inexpensive tool for quantitative proteome analysis in thyroid nodular disease. .COPYRGT. 2004 Elsevier Ireland Ltd. All rights reserved.

L9 ANSWER 13 OF 37 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:591440 HCAPLUS
DOCUMENT NUMBER: 139:148006
TITLE: Comparative gene profiling between undifferentiated and well-differentiated nasopharyngeal carcinoma (NPC) cells for NPC diagnosis and treatment
INVENTOR(S): Ng, Aylwin; Tang, Jing P.; Hui, Kam M.; Goh, Christopher H. K.
PATENT ASSIGNEE(S): Biotech Research Ventures Pte Limited, Singapore; Cripps, Joanna E.
SOURCE: PCT Int. Appl., 61 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003062826	A2	20030731	WO 2003-GB329	20030123
WO 2003062826	A3	20031016		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CN 1643377	A	20050720	CN 2003-806030	20030123
PRIORITY APPLN. INFO.:			GB 2002-1498 A	20020123

AB The invention is concerned with the detection and treatment of nasopharyngeal carcinoma (NPC) based on differential gene **expression** in these cells. Specifically, the invention provides details of differentially **expressed** genes in NPC which serve to detect the presence or risk of the disease and its clin. type. A set of genes are identified by DNA microarray using comparative gene profiling between undifferentiated and well-differentiated nasopharyngeal carcinoma (NPC) cells (CNE-2 or HK1 resp.) to be associated with NPC, in particular, two imprinted genes H19 (no protein product, function unknown) and CDKN1C (cyclin-dependent kinase inhibitor 1C) located on chromosome 11p15. It is also shown H19 gene is highly **expressed** in CNE-2 cells and hypermethylation of its CpG promoter region is detected in the HK1 cells. Furthermore, hypomethylation of the CpG dinucleotides within H19 promoter region is correlated with the restoration of its mRNA **expression** in HK1 cells. The invention also provides methods of treating NPC in association with chemo or radiotherapy.

L9 ANSWER 14 OF 37 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:334399 HCAPLUS
DOCUMENT NUMBER: 138:352748
TITLE: WT1 protein-derived immunogenic peptides for immunotherapy of leukemia and cancer
INVENTOR(S): Gaiger, Alexander; Cheever, Martin A.; McNeill,

Patricia D.; Smithgall, Molly; Moulton, Gus; Vedvick, Thomas S.; Sleath, Paul R.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 161 pp., Cont.-in-part of U. S. Ser. No. 685,830.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 11
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2003082196	A1	20030501	US 2001-785019	20010215
US 2003072767	A1	20030417	US 2001-938864	20010824
CA 2425072	AA	20020411	CA 2001-2425072	20011003
WO 2002028414	A1	20020411	WO 2001-US31139	20011003
WO 2002028414	B1	20020718		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2001096608	A5	20020415	AU 2001-96608	20011003
EP 1328287	A1	20030723	EP 2001-977493	20011003
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
JP 2004510425	T2	20040408	JP 2002-532238	20011003
CN 1505526	A	20040616	CN 2001-819114	20011003
US 2003095971	A1	20030522	US 2001-2603	20011030
US 2003039635	A1	20030227	US 2002-125635	20020416
US 2003198622	A1	20031023	US 2002-195835	20020712
US 2003235557	A1	20031225	US 2002-244830	20020916
US 2003215458	A1	20031120	US 2002-286333	20021030
US 2004126362	A1	20040701	US 2003-648780	20030826
AU 2003257511	A1	20031120	AU 2003-257511	20031023

PRIORITY APPLN. INFO.:

US 2000-685830	A2	20001009
US 1998-164223	A2	19980930
US 1999-276484	A2	19990325
AU 1999-64078	A3	19990930
US 2000-684361	A2	20001006
US 2001-785019	A2	20010215
US 2001-938864	A	20010824
WO 2001-US31139	W	20011003
US 2001-2603	A2	20011030
US 2002-125635	A2	20020416
US 2002-195835	A2	20020712
US 2002-244830	A2	20020916

AB Compns. and methods for the therapy of malignant diseases, such as leukemia and cancer, are disclosed. The compns. comprise one or more of a WT1 polynucleotide, a WT1 polypeptide, an antigen-presenting cell presenting a WT1 polypeptide, an antibody that specifically binds to a WT1 polypeptide; or a T cell that specifically reacts with a WT1 polypeptide. Such compns. may be used, for example, for the prevention and treatment of metastatic diseases.

L9 ANSWER 15 OF 37 MEDLINE on STN
ACCESSION NUMBER: 2003126656 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12514184
TITLE: Catalytic mechanism of thiol peroxidase from Escherichia

coli. Sulfenic acid formation and overoxidation of essential CYS61.
AUTHOR: Baker Laura M S; Poole Leslie B
CORPORATE SOURCE: Department of Biochemistry, Wake Forest University School of Medicine, Winston-Salem, North Carolina 27157, USA.
CONTRACT NUMBER: R01 GM50389 (NIGMS)
SOURCE: The Journal of biological chemistry, (2003 Mar 14) Vol. 278, No. 11, pp. 9203-11. Electronic Publication: 2003-01-03.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200305
ENTRY DATE: Entered STN: 20030319
Last Updated on STN: 20030515
Entered Medline: 20030514

AB Escherichia coli thiol peroxidase (Tpx, p20, scavengase) is part of an oxidative stress defense system that uses reducing equivalents from **thioredoxin** (Trx1) and **thioredoxin** reductase to reduce alkyl hydroperoxides. Tpx contains three Cys residues, Cys(95), Cys(82), and Cys(61), and the latter residue aligns with the N-terminal active site Cys of other peroxidases in the peroxiredoxin family. To identify the catalytically important Cys, we have **cloned** and purified Tpx and four mutants (C61S, C82S, C95S, and C82S,C95S). In rapid reaction kinetic experiments measuring steady-state turnover, C61S is inactive, C95S retains partial activity, and the C82S mutation only slightly affects reaction rates. Furthermore, a sulfenic acid intermediate at Cys(61) generated by cumene hydroperoxide (CHP) treatment was detected in UV-visible spectra of 4-nitrobenzo-2-oxa-1,3-diazole-labeled C82S,C95S, confirming the identity of Cys(61) as the peroxidatic center. In stopped-flow kinetic studies, Tpx and Trx1 form a Michaelis complex during turnover with a catalytic efficiency of 3.0 x 10(6) m(-1) s(-1), and the low K(m) (9.0 microm) of Tpx for CHP demonstrates substrate specificity toward alkyl hydroperoxides over H(2)O(2) (K(m) > 1.7 mm). Rapid inactivation of Tpx due to Cys(61) overoxidation is observed during turnover with CHP and a lipid hydroperoxide, 15-hydroperoxyeicosatetraenoic acid, but not H(2)O(2). Unlike most other 2-Cys peroxiredoxins, which operate by an intersubunit disulfide mechanism, Tpx contains a redox-active intrasubunit disulfide bond yet is homodimeric in solution.

L9 ANSWER 16 OF 37 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:25886 BIOSIS
DOCUMENT NUMBER: PREV200400024278
TITLE: PROBIOTICS CHANGE E. COLI-INDUCED GENE **EXPRESSION** PROFILES IN CACO-2 CELLS.
AUTHOR(S): Panigrahi, Pinaki [Reprint Author]; Braileanu, Gheorghe [Reprint Author]
CORPORATE SOURCE: Baltimore, MD, USA
SOURCE: Digestive Disease Week Abstracts and Itinerary Planner, (2003) Vol. 2003, pp. Abstract No. T1082. e-file.
Meeting Info.: Digestive Disease 2003. FL, Orlando, USA. May 17-22, 2003. American Association for the Study of Liver Diseases; American Gastroenterological Association; American Society for Gastrointestinal Endoscopy; Society for Surgery of the Alimentary Tract.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 31 Dec 2003

Last Updated on STN: 31 Dec 2003

AB Background: Lactobacillus group of organisms (probiotics) are known for their protective role in infectious and inflammatory conditions of the intestine. In our previous studies, Lactobacillus plantarum (Lp) has been shown to block E. coli-induced tissue injury in a weanling rabbit ileal loop model via modulation of inflammatory response. We have further reported the **expression** of specific genes including enterocyte differentiation factor, aquaporin 3, nucleoporin, oxidoreductase and **thioredoxin** reductase 2 by Lp in Caco-2 cells. Aim: In this study, DNA microarray technique was used to examine the induction of genes after infection with (i) E. coli, (ii) Lp, and (iii) the combination (E. coli+Lp in co-culture) in cultured Caco-2 cells.. Methods: Differentiated Caco2 cells were treated for 2 hr with E. coli (109 CFU/ml), Lp (109 CFU/ml) or the combination. Total RNA was extracted using the Trizol method. A 3DNA submicro labeling kit for 2 step protocol (Genisphere) was used for hybridization in a glass microarray with 19 000 human ESTs/genes in duplicate (University of Toronto). Samples (control and three experimental conditions) were labeled with Alexa 488, 546 594 and 647 **dyes**, scanned with 4 different lasers, and analyzed (including hierarchial cluster analysis) using the ImaGene 4.2. and GeneSight 3 (Biodiscovery Inc.) softwares. Results: A set of 323, 347 and 262 genes/ESTs were induced by E. coli, Lp and the combination respectively. Interestingly, of these genes, while E. coli and Lp stimulated 203 and 221 genes respectively, the co-infection resulted in only the stimulation of 160 genes. E. coli and LP suppressed 120, and 126 genes respectively with only 102 genes suppressed during mixed infection. Of the 323 and 262 genes **expressed** during E. coli and mixed infection, 170 gene were found to be common, and there were 153 and 92 unique genes in these two conditions. Further analysis of these genes/ESTs are currently underway in our laboratory. Conclusions: Although **expression** of different sets of genes was expected by specific infections in this experiment, it was intriguing to note that addition of Lp changed (reduced) the primary genes induced by E. coli alone. The phenomenon of 'reversal' of activation and repression (by E. coli) of gene sets by Lp as observed in this study may point to the inherent ability of probiotics in affecting eukaryotic genes and thereby modulating the infective and inflammatory events induced by normal flora and pathogenic bacteria in the intestine..

L9 ANSWER 17 OF 37 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2002:267906 BIOSIS
DOCUMENT NUMBER: PREV200200267906
TITLE: Molecular characterization of a novel protein disulfide isomerase in carrot.
AUTHOR(S): Xu, Zheng-Jun; Ueda, Kenji; Masuda, Kiyoshi; Ono, Michiyuki; Inoue, Masayasu [Reprint author]
CORPORATE SOURCE: Biotechnology Institute, Akita Prefectural University, Ohgata, Akita, 010-0444, Japan
ino@agri.akita-pu.ac.jp
SOURCE: Gene (Amsterdam), (6 February, 2002) Vol. 284, No. 1-2, pp. 225-231. print.
CODEN: GENED6. ISSN: 0378-1119.
DOCUMENT TYPE: Article
LANGUAGE: English
OTHER SOURCE: DDBJ-D85891
ENTRY DATE: Entered STN: 1 May 2002
Last Updated on STN: 1 May 2002

AB A protein disulfide isomerase (PDI) coding sequence was **cloned** from a cDNA library derived from carrot (Daucus carota L.) somatic embryos. The cDNA is 2060 bp in length and encodes for a protein of 581 amino acids and molecular weight of 64.4 kDa. Primary structure analysis of the deduced protein revealed two **thioredoxin**-like active sites and an endoplasmic reticulum-retention signal at its C-terminus,

which is also found in PDIs in plants and animals. Although between the carrot protein and other plant PDIs there is only about 30% identity, the active site regions are almost identical. The corresponding mRNA was found in varying amounts, in all tissues investigated. A **recombinant** protein **expressed** from the carrot cDNA **clone** effectively catalyzed both glutathione-insulin transhydrogenation and the oxidative renaturation of denatured RNase A. These results suggest that the protein coded for by the carrot gene is a novel member of the PDI family in plants. We therefore designated this novel carrot gene PDIL1. The protein **expressed** by the PDIL1 cDNA sequence had a highly acidic stretch at its N-terminal region (no such domain exists in known plant PDIs), and was located far from known plant PDIs on a maximum likelihood tree. The PDIL1 gene, together with closely-related genes identified in Arabidopsis and tomato, was suggested to belong to a novel subfamily of PDIs.

L9 ANSWER 18 OF 37 MEDLINE on STN
ACCESSION NUMBER: 2002094790 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11792180
TITLE: Poly[Lys-(AEDTP)]: a cationic polymer that allows dissociation of pDNA/cationic polymer complexes in a reductive medium and enhances polyfection.
AUTHOR: Pichon Chantal; LeCam Eric; Guerin Brigitte; Coulaud Dominique; Delain Etienne; Midoux Patrick
CORPORATE SOURCE: Centre de Biophysique Moleculaire, CNRS UPR 4301, rue Charles Sadron, F-45071 Orleans Cedex 02, France.
SOURCE: Bioconjugate chemistry, (2002 Jan-Feb) Vol. 13, No. 1, pp. 76-82.
Journal code: 9010319. ISSN: 1043-1802.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200204
ENTRY DATE: Entered STN: 20020205
Last Updated on STN: 20020406
Entered Medline: 20020405

AB Polyplexes of high stability resulting from the condensation of a plasmid DNA by a cationic polymer are widely used to develop polymer-based gene delivery systems. However, the plasmid must be released from its vector once inside the cells for an efficient **expression** of the exogenous gene in the cell nucleus. We have designed a disulfide-containing cationic polymer termed poly[Lys-(AEDTP)] which allowed for the formation of polyplexes and the release of the plasmid in a reductive medium. The amino groups of polylysine were substituted with 3-(2-aminoethyldithio)propionyl residues in order to have each amino group of poly[Lys-(AEDTP)] interacting with a phosphate DNA linked to the polymer backbone via a disulfide bond. As evidenced by agarose gel electrophoresis and ethidium bromide/pDNA fluorescence restoration, poly[Lys-(AEDTP)] polyplexes were decondensed and the plasmid released upon treatment with either dithiothreitol, glutathione in the presence of glutathione reductase, or the **thioredoxin** reductase. Electron microscopy showed that polyplexes exhibiting spherical particles of a mean size at about 100 nm were decondensed in the presence of glutathione and exhibited filamentous aggregates. Finally, we found that the transfection of 293T7 and HepG2 cells was 10- and 50-fold more efficient with poly[Lys-(AEDTP)] polyplexes, respectively, than with poly[Lys] polyplexes. These results indicate that disulfide-containing cationic polymers must be borne in mind for developing polymer-base gene delivery systems.

L9 ANSWER 19 OF 37 NTIS COPYRIGHT 2006 NTIS on STN
ACCESSION NUMBER: 2001(16):04701
NTIS ORDER NUMBER: ADA388146/XAB

TITLE: Biomimetic Sensor for Pathogenic Bacteria. Final rept 1 May-Sep 30 2000.
AUTHOR: Walsh, M. K.; De Wald, D. B.; Weimer, B. C.
CORPORATE SOURCE: Utah State Univ., Logan. Dept. of Nutrition and Food Science. (004748080 407359)
NUMBER OF REPORT: ADA388146/XAB; AFRL; SR-BL-TR-01-0190
17p; 5 Mar 2001
NUMBER OF CONTRACT: F49620-00-1-0289
2312
DX
CONTROLLED TERM: Report
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: Product reproduced from digital image. Order this product from NTIS by: phone at 1-800-553-NTIS (U.S. customers); (703)605-6000 (other countries); fax at (703)605-6900; and email at orders@ntis.gov. NTIS is located at 5285 Port Royal Road, Springfield, VA, 22161, USA.
NTIS Prices: PC A03/MF A01
OTHER SOURCE: GRA&I0115

AB This study investigated the assembly of a biomimetic sensor containing an osmotic receptor protein. The first objective of this research included the assembly and immobilization of fluorescently tagged liposomes. The second objective involved the **expression** and purification of an osmotic sensitive protein (MscL) and the incorporation of this protein into the liposome membrane. Liposomes (2 micron diameter) containing fluorescein labeled phospholipids and biotinylated phosphotidyl ethanolamine in the membranes and internalized soluble sulforhodamine were assembled. Liposomes were characterized with respect to composition, size, and shelf-life using confocal microscopy. Avidin was covalently attached to a glass surface for the immobilization of the biotinylated liposomes. Immobilization of fluorescent liposomes was confirmed with confocal microscopy. The liposomes contain a green/yellow lipid bilayer and a red interior. The **cloning** of **recombinant** MScL into an Escherichia coli **expression** system yielded an MScL-**thioredoxin** fusion protein that was tagged with a blue fluorescent **dye** and incorporated into the membrane of the liposomes. The functionality of the proteosome was observed by the release of the water soluble sulforhodamine in the presence of high salt, 3 M, concentrations.

L9 ANSWER 20 OF 37 MEDLINE on STN
ACCESSION NUMBER: 2001518508 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11565801
TITLE: Proteomic characterization of early-stage differentiation of mouse embryonic stem cells into neural cells induced by all-trans retinoic acid in vitro.
AUTHOR: Guo X; Ying W; Wan J; Hu Z; Qian X; Zhang H; He F
CORPORATE SOURCE: Department of Genomics and Proteomics, Beijing Institute of Radiation Medicine, China.
SOURCE: Electrophoresis, (2001 Aug) Vol. 22, No. 14, pp. 3067-75. Journal code: 8204476. ISSN: 0173-0835.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200204
ENTRY DATE: Entered STN: 20010924
Last Updated on STN: 20020409
Entered Medline: 20020408

AB Embryonic stem (ES) cells are totipotent stem cells, which can differentiate into various kinds of cell types, including neurons. They are widely used as a model system for investigating mechanisms of

differentiation events during early mouse development. In this study, proteomic techniques were used to approach the protein profile associated with the early-stage differentiation of ES cells into neuronal cells induced by all-trans retinoic acid (ATRA) in vitro. In comparison of the protein profile of parent ES cells with that of ES-derived neural-committed cells, which was induced by ATRA for four days, 24 differentially displayed protein spots were selected from two-dimensional electrophoresis (2-DE) gels for further protein identification by pepide mass fingerprinting (PMF). Nine proteins were known to being involved in the process of neural differentiation and/or neural survival. Of those, alpha-3/alpha-7 tubulin and vimentin were down-regulated, while cytokeratin 8, cytokeratin 18, G1/S-special cyclin D2, follistatin-related protein, NEL protein, platelet-activating factor acetylhydrolase IB alpha-subunit, and **thioredoxin** peroxidase 2 were upregulated during differentiation of ES cells to neural cells. Additionally, other 12 protein (five upregulated and seven downregulated) spots associated with ES cell differentiation into neuronal cells were not matched to known proteins so far, implicating that they might be novel proteins. The results above indicated that the molecular mechanisms of differentiation of ES cells to neural cells in vitro might be similar to those of other neural systems in vitro and identified that proteomic analysis is an effective strategy to comprehensively unravel the regulatory network of differentiation.

L9 ANSWER 21 OF 37 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2001:98372 HCAPLUS
DOCUMENT NUMBER: 134:232542
TITLE: Genome sequence of enterohaemorrhagic Escherichia coli O157:H7
AUTHOR(S): Perna, Nicole T.; Plunkett, Guy, III; Burland, Valerie; Mau, Bob; Glasner, Jeremy D.; Rose, Debra J.; Mayhew, George F.; Evans, Peter S.; Gregor, Jason; Kirkpatrick, Heather A.; Posfai, Gyorgy; Hackett, Jeremiah; Klink, Sara; Boutin, Adam; Shao, Ying; Miller, Leslie; Grotbeck, Erik J.; Davis, N. Wayne; Lim, Alex; Dimalanta, Eileen T.; Potamousis, Konstantinos D.; Apodaca, Jennifer; Anantharaman, Thomas S.; Lin, Jieyi; Yen, Glaex; Schwartz, David C.; Welch, Rodney A.; Blattner, Frederick R.
CORPORATE SOURCE: Genome Center of Wisconsin, Department of Animal Health and Biomedical Sciences, Laboratory of Genetics, Department of Chemistry, Department of Biostatistics, and Department of Medical Microbiology and Immunology, University of Wisconsin, Madison, WI, 53706, USA
SOURCE: Nature (London) (2001), 409(6819), 529-533
CODEN: NATUAS; ISSN: 0028-0836
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The bacterium Escherichia coli O157:H7 is a worldwide threat to public health and has been implicated in many outbreaks of hemorrhagic colitis, some of which included fatalities caused by hemolytic uremic syndrome. Close to 75,000 cases of O157:H7 infection are now estimated to occur annually in the United States. The severity of disease, the lack of effective treatment and the potential for large-scale outbreaks from contaminated food supplies have propelled intensive research on the pathogenesis and detection of E. coli O157:H7. The genome of E. coli O157:H7 was sequenced to identify candidate genes responsible for pathogenesis, to develop better methods of strain detection and to advance our understanding of the evolution of E. coli, through comparison with the genome of the non-pathogenic laboratory strain E. coli K-12. Lateral gene transfer found to be far more extensive than previously anticipated. In fact, 1387 new genes encoded in strain-specific clusters of diverse sizes were found in

O157:H7. These include candidate virulence factors, alternative metabolic capacities, several prophages, and other new functions - all of which could be targets for surveillance.

REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 22 OF 37 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2001:209011 HCAPLUS
DOCUMENT NUMBER: 137:196299
TITLE: Genome sequence of enterohaemorrhagic Escherichia coli O157:H7. [Erratum to document cited in CA134:232542]
AUTHOR(S): Perna, Nicole T.; Plunkett, Guy, III; Burtand, Valerie; Mau, Bob; Glasner, Jeremy D.; Rose, Debra J.; Mayhew, George F.; Evans, Peter S.; Gregor, Jason; Kirkpatrick, Heather A.; Postal, Gyorgy; Hackett, Jeremiah; Klink, Sara; Boutin, Adam; Shao, Ying; Miller, Leslie; Grotbeck, Erik J.; Davis, N. Wayne; Lim, Alex; Dimalanta, Eileen T.; Potamousis, Konstantinos D.; Apodaca, Jennifer; Anantharaman, Thomas S.; Lin, Jleyl; Yen, Galex; Schwartz, Dvaid C.; Welch, Rodney A.; Blatner, Frederick R.
CORPORATE SOURCE: Genome Center of Wisconsin, Department of Animal Health and Biomedical Sciences, Laboratory of Genetics, Department of Chemistry, Department of Biostatistics, and Department of Medical Microbiology and Immunology, University of Wisconsin, Madison, WI, 53706, USA
SOURCE: Nature (London, United Kingdom) (2001), 410(6825), 240
CODEN: NATUAS; ISSN: 0028-0836
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The correct GenBank accession number for the annotated sequence is AE005174.

L9 ANSWER 23 OF 37 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2001:209816 BIOSIS
DOCUMENT NUMBER: PREV200100209816
TITLE: Fluorescently labeled model DNA sequences for exonucleolytic sequencing.
AUTHOR(S): Foeldes-Papp, Zeno [Reprint author]; Angerer, Bernhard; Thyberg, Per; Hinz, Michael; Wennmalm, Stefan; Ankenbauer, Waltraud; Seliger, Hartmut; Holmgren, Arne; Rigler, Rudolf
CORPORATE SOURCE: Clinical Immunology and Jean Dausset Laboratory, Graz University M.S. and Hospital, Auenbrugger Platz 8, A-8036, Graz, Austria
Zeno.Foldes-Papp@kfunigraz.ac.at
SOURCE: Journal of Biotechnology, (13 April, 2001) Vol. 86, No. 3, pp. 203-224. print.
CODEN: JBITD4. ISSN: 0168-1656.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 2 May 2001
Last Updated on STN: 18 Feb 2002
AB We describe here the enzyme-catalyzed, low-density labeling of DNAs with fluorescent **dyes**. Firstly, for 'natural' template DNAs, dNTPs were partially substituted in the labeling reactions by the respective fluorophore-bearing analogs. The DNAs were labeled by PCR using Taq DNA polymerase. The covalent incorporation of **dye**-dNTPs decreased in the following order: rhodamine-green-5-dUTP (Molecular Probes, the Netherlands), tetramethylrhodamine-4-dUTP (FluoroRed, Amersham Pharmacia Biotech), Cy5-dCTP (Amersham Pharmacia Biotech). Exonucleolytic degradation by the 3' fwdarw 5' exonuclease activity of T7 DNA polymerase (wild type) in the presence of excess reduced **thioredoxin**

proceeded to complete breakdown of the labeled DNAs. The catalytic cleavage constants determined by fluorescence correlation spectroscopy were between 0.5 and 1.5 s-1 at 16degreeC, normalized for the covalently incorporated dye-nucleotides. Secondly, rhodamine-green-X-dUTP (Roche Diagnostics), tetramethylrhodamine-6-dUTP (Roche Diagnostics), and Cy5-dCTP were covalently incorporated into the antisense strand of 'synthetic' 218-b DNA template constructs (master sequences) at well defined positions, starting from the primer binding site, by total substitution for the naturally occuring dNTPs. The 218-b DNA constructs were labeled by PCR with a thermostable 3' fwdarw 5' exonuclease deficient mutant of the Tgo DNA polymerase which we have selected. The advantage of the special, synthetic DNA constructs as compared to natural DNAs lies in the possibility of obtaining tailor-made nucleic acids, optimized for testing the performance of exonucleolytic sequencing. The number of incorporated fluorescent nucleotides determined by complete exonucleolytic degradation and fluorescence correlation spectroscopy were six out of six possible incorporations for rhodamine-green-X-dUTP and tetramethylrhodamine-6-dUTP, respectively. Their covalent and base-specific incorporations were confirmed by the novel analysis methodology of re-sequencing (i.e. mobility-shift gel electrophoresis, reversion-PCR and re-sequencing) first developed in the paper Foldes-Papp et al. (2001) and in this paper. This methodology was then used by other groups within the whole sequencing project.

L9 ANSWER 24 OF 37 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2001:184271 HCAPLUS
DOCUMENT NUMBER: 134:217892
TITLE: Complete genome sequence of enterohemorrhagic Escherichia coli O157:H7 and genomic comparison with a laboratory strain K-12
AUTHOR(S): Hayashi, Tetsuya; Makino, Kozo; Ohnishi, Makoto; Kurokawa, Ken; Ishii, Kazuo; Yokoyama, Katsushi; Han, Chang-Gyun; Ohtsubo, Eiichi; Nakayama, Keisuke; Murata, Takahiro; Tanaka, Masashi; Tobe, Toru; Iida, Tetsuya; Takami, Hideto; Honda, Takeshi; Sasakawa, Chihiro; Ogasawara, Naotake; Yasunaga, Teruo; Kuhara, Satoru; Shiba, Tadayoshi; Hattori, Masahira; Shinagawa, Hideo
CORPORATE SOURCE: Department of Microbiology, Miyazaki Medical College, Miyazaki, 899-1692, Japan
SOURCE: DNA Research (2001), 8(1), 11-22
CODEN: DARSE8; ISSN: 1340-2838
PUBLISHER: Universal Academy Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Escherichia coli O157:H7 is a major food-borne infectious pathogen that causes diarrhea, hemorrhagic colitis, and hemolytic uremic syndrome. The complete chromosome sequence of an O157:H7 strain isolated from the Sakai outbreak is reported, and the results compared with the genome of a benign laboratory strain, K-12 MG1655. The chromosome is 5.5 Mb in size, 859 Kb larger than that of K-12. A 4.1-Mb sequence highly conserved between the two strains is identified, which may represent the fundamental backbone of the E. coli chromosome. The remaining 1.4-Mb sequence comprises of O157:H7-specific sequences, most of which are horizontally transferred foreign DNAs. The predominant roles of bacteriophages in the emergence of O157:H7 is evident by the presence of 24 prophages and prophage-like elements that occupy more than half of the O157:H7-specific sequences. The O157:H7 chromosome encodes 1632 proteins and 20 tRNAs that are not present in K-12. Among these, at least 131 proteins are assumed to have virulence-related functions. Genome-wide codon usage anal. suggested that the O157:H7-specific tRNAs are involved in the efficient **expression** of the strain-specific genes. A complete set of the genes specific to O157:H7 presented here sheds new insight into the

pathogenicity and the physiol. of O157:H7, and will open a way to fully understand the mol. mechanisms underlying the O157:H7 infection.

REFERENCE COUNT: 62 THERE ARE 62 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 25 OF 37 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2000:666972 HCAPLUS
DOCUMENT NUMBER: 133:219795
TITLE: Proteins for diagnosis and treatment of breast cancer
INVENTOR(S): Amess, Bob; Townsend, Robert Reid; Parekh, Rajesh Bhikhu; Waterfield, Michael Derek; O'Hare, Michael John
PATENT ASSIGNEE(S): Oxford Glycosciences (UK) Ltd., UK
SOURCE: PCT Int. Appl., 86 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000055628	A1	20000921	WO 2000-GB908	20000313
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1159618	A1	20011205	EP 2000-909494	20000313
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				

PRIORITY APPLN. INFO.: GB 1999-5817 A 19990312
WO 2000-GB908 W 20000313

AB The present invention provides methods and compns. for screening, diagnosis and prognosis of breast cancer, for monitoring the effectiveness of breast cancer treatment, and for drug development. Breast Cancer-Associated Features (BFs), detectable by two-dimensional electrophoresis of breast tissue, are described. The invention further provides Breast Cancer-Associated Protein Isoforms (BPIs) detectable in breast tissue, prepns. comprising isolated BPIs, antibodies immunospecific for BPIs, and kits comprising the aforesaid. Luminal and myoepithelial cells were purified by immunomagnetic methods from 10 sets of matched normal and cancer breast cell tissue. Two-dimensional electrophoresis was used to sep. the proteins. High resolution detection of protein features using fluorescent **dyes**, coupled to advanced software to identify differentially **expressed** features, high through-put mass spectrometry and bioinformatics was also applied. This has allowed the identification of large sets of proteins which are differentially **expressed** between the luminal and myoepithelial human breast cell proteomes.

REFERENCE COUNT: 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 26 OF 37 MEDLINE on STN
ACCESSION NUMBER: 2000290849 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10828978
TITLE: AhpF can be dissected into two functional units: tandem repeats of two **thioredoxin**-like folds in the N-terminus mediate electron transfer from the **thioredoxin** reductase-like C-terminus to AhpC.

AUTHOR: Poole L B; Godzik A; Nayeem A; Schmitt J D
CORPORATE SOURCE: Department of Biochemistry, Wake Forest University School of Medicine, Winston-Salem, North Carolina 27157, USA.. lbpoole@wfubmc.edu
CONTRACT NUMBER: GM50389 (NIGMS)
GM60049 (NIGMS)
SOURCE: Biochemistry, (2000 Jun 6) Vol. 39, No. 22, pp. 6602-15.
Journal code: 0370623. ISSN: 0006-2960.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200007
ENTRY DATE: Entered STN: 20000720
Last Updated on STN: 20021210
Entered Medline: 20000710

AB AhpF, the flavin-containing component of the Salmonella typhimurium alkyl hydroperoxide reductase system, catalyzes the NADH-dependent reduction of an active-site disulfide bond in the other component, AhpC, which in turn reduces hydroperoxide substrates. The amino acid sequence of the C-terminus of AhpF is 35% identical to that of **thioredoxin** reductase (TrR) from Escherichia coli. AhpF contains an additional 200-residue N-terminal domain possessing a second redox-active disulfide center also required for AhpC reduction. Our studies indicate that this N-terminus contains a tandem repeat of two **thioredoxin** (Tr)-like folds, the second of which contains the disulfide redox center. Structural and catalytic properties of independently **expressed** fragments of AhpF corresponding to the TrR-like C-terminus (F[208-521]) and the 2Tr-like N-terminal domain (F[1-202]) have been addressed. Enzymatic assays, reductive titrations, and circular dichroism studies of the fragments indicate that each folds properly and retains many functional properties. Electron transfer between F[208-521] and F[1-202] is, however, relatively slow (4 x 10(4) M(-)(1) s(-)(1) at 25 degrees C) and nonsaturable up to 100 microM F[1-202]. TrR is nearly as efficient at F[1-202] reduction as is F[208-521], although neither the latter fragment, nor intact AhpF, can reduce Tr. An engineered mutant AhpC substrate with a fluorophore attached via a disulfide bond has been used to demonstrate that only F[1-202], and not F[208-521], is capable of electron transfer to AhpC, thereby establishing the direct role this N-terminal domain plays in mediating electron transfer between the TrR-like part of AhpF and AhpC.

L9 ANSWER 27 OF 37 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1999:747450 HCAPLUS
DOCUMENT NUMBER: 132:1816
TITLE: A protein-labeling compound, and a method for labeling protein using this compound
INVENTOR(S): Yanagawa, Hiroshi; Nemoto, Naoto; Miyamoto, Etsuko
PATENT ASSIGNEE(S): Mitsubishi Chemical Industries Ltd., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 9 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 11322781	A2	19991124	JP 1998-133170	19980515
US 6228994	B1	20010508	US 1998-190276	19981113
US 2001039011	A1	20011108	US 2000-737751	20001218
US 2001007751	A1	20010712	US 2001-794128	20010228

PRIORITY APPLN. INFO.: JP 1998-133170 A 19980515
US 1998-190276 A3 19981113

AB A protein-labeling compound is chemical synthesized from puromycin or puromycin

analog and a functional compound by forming a chemical bond between them. This protein-labeling compound is made of a labeling part consisting of a labeling chemical and an acceptor part consisting of a chemical capable of binding to C-terminus of a protein. A labeling of a protein is carried out by adding the protein-labeling compound at the final concentration of 10-0.01μM to the translation system in cell-free extract of prokaryotic or eukaryotic cell, or to living cells. A labeling part chemical can be one of various functional compds. such as a fluorescent **dye** and a radioactive substance. The protein-labeling compound is useful in detecting or identifying the protein **expressed** in various translation systems of cell-free extract or living cells. The identification of the corresponding proteins is the most important task for the functional anal. of the genes which have been accumulated by genome anal. An extremely effective means is provided by this type of protein-labeling compound from the stand point of an efficiency or automation in the functional anal. of proteins such as nucleic acid-protein interaction or protein-protein interaction. As examples, ribocytidyl(3'→5')puromycin, deoxyuridyl(3'→5')puromycin, deoxycytidyl(3'→5')puromycin, Fluorpur and Fluorthiopur were synthesized and effectively used for the labeling of **thioredoxin expressed** in E. coli cell-free extract

L9 ANSWER 28 OF 37 BIOTECHDS COPYRIGHT 2006 THE THOMSON CORP. on STN
DUPLICATE 1
ACCESSION NUMBER: 1998-09126 BIOTECHDS
TITLE: Production of polypeptides as inclusion bodies; **recombinant** protein preparation by plasmid pTrc99A or plasmid pTrxfus vector-mediated **thioredoxin expression** in Escherichia coli inclusion body
AUTHOR: Chatterjee D; Longo M; Flynn E; Oberfelder R
PATENT ASSIGNEE: Life-Technol.
LOCATION: Rockville, MD, USA.
PATENT INFO: WO 9830684 16 Jul 1998
APPLICATION INFO: WO 1998-US492 8 Jan 1998
PRIORITY INFO: US 1997-34658 8 Jan 1997
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 1998-399134 [34]
AB A new method for the preparation of a protein in the form of an inclusion body involves: obtaining a host cell (e.g. Escherichia coli) containing a first DNA sequence encoding the protein linked to a second DNA sequence encoding an inclusion partner (**thioredoxin** or modified **thioredoxin**), forming a gene fusion construct (plasmid pTrcpr1-monomer or pTrxA-concat); and culturing the cell to favor production of the protein as inclusion bodies. Also new are: vector plasmid pTrc99A and plasmid pTrxfus containing the construct; a host cell containing the vector; making a protein mol.weight ladder composition by obtaining one or more DNA sequences encoding proteins of different mol.weight values, transforming host cells with the DNA, culturing the cells to favor production of each protein, and isolating each protein; and making one or more stained proteins by incubating the proteins with one or more protein-binding **dyes** under incubation conditions to complex the proteins with the **dyes**. The methods may be used to prepare a fragment of the gene-32 protein of phage T4, a fragment of KpnI-methylase or a fragment of E. coli Dead-Box protein or **thioredoxin**. (84pp)

L9 ANSWER 29 OF 37 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 1998288331 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9624185
TITLE: Disruption of redox homeostasis in the transforming growth factor-alpha/c-myc transgenic mouse model of accelerated hepatocarcinogenesis.

AUTHOR: Factor V M; Kiss A; Woitach J T; Wirth P J; Thorgeirsson S S
CORPORATE SOURCE: Laboratory of Experimental Carcinogenesis, NCI, National Institutes of Health, Bethesda, Maryland 20892, USA.
SOURCE: The Journal of biological chemistry, (1998 Jun 19) Vol. 273, No. 25, pp. 15846-53.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199807
ENTRY DATE: Entered STN: 19980716
Last Updated on STN: 19980716
Entered Medline: 19980709

AB In previous studies we have demonstrated that transforming growth factor (TGF)-alpha/c-myc double transgenic mice exhibit an enhanced rate of cell proliferation, accumulate extensive DNA damage, and develop multiple liver tumors between 4 and 8 months of age. To clarify the biochemical events that may be responsible for the genotoxic and carcinogenic effects observed in this transgenic model, several parameters of redox homeostasis in the liver were examined prior to development of hepatic tumors. By 2 months of age, production of reactive oxygen species, determined by the peroxidation-sensitive fluorescent **dye**, 2',7'-dichlorofluorescin diacetate, was significantly elevated in TGF-alpha/c-myc transgenic hepatocytes versus either wild type or c-myc single transgenic cells, and occurred in parallel with an increase in lipid peroxidation. Concomitantly with a rise in oxidant levels, antioxidant defenses were decreased, including total glutathione content and the activity of glutathione peroxidase, whereas **thioredoxin** reductase activity was not changed. However, hepatic tumors which developed in TGF-alpha/c-myc mice exhibited an increase in **thioredoxin** reductase activity and a very low activity of glutathione peroxidase. Furthermore, specific deletions were detected in mtDNA as early as 5 weeks of age in the transgenic mice. These data provide experimental evidence that co-**expression** of TGF-alpha and c-myc transgenes in mouse liver promotes overproduction of reactive oxygen species and thus creates an oxidative stress environment. This phenomenon may account for the massive DNA damage and acceleration of hepatocarcinogenesis observed in the TGF-alpha/c-myc mouse model.

L9 ANSWER 30 OF 37 MEDLINE on STN
ACCESSION NUMBER: 1998181877 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9523703
TITLE: Characterization of wheat **thioredoxin** h cDNA and production of an active Triticum aestivum protein in Escherichia coli.
AUTHOR: Gautier M F; Lullien-Pellerin V; de Lamotte-Guery F; Guirao A; Joudrier P
CORPORATE SOURCE: Unite de Biochimie et Biologie Moleculaire des Cereales, INRA, Montpellier, France.. gautier@ensam.inra.fr
SOURCE: European journal of biochemistry / FEBS, (1998 Mar 1) Vol. 252, No. 2, pp. 314-24.
Journal code: 0107600. ISSN: 0014-2956.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AJ001903; GENBANK-X69915
ENTRY MONTH: 199804
ENTRY DATE: Entered STN: 19980430
Last Updated on STN: 20000303
Entered Medline: 19980423

AB Two cDNA **clones**, pTaM13.38 and pTd14.13.2, encoding a Triticum

aestivum and a Triticum durum **thioredoxin** h, respectively, were isolated from mid-maturation seed cDNA libraries. The T aestivum **thioredoxin** h has a molecular mass of 13.5 kDa and that from T durum has a molecular mass of 13.8 kDa. These two wheat **thioredoxin** h are 98.5% similar and contain the canonical WCGPC active site and the important structural and functional amino acids that are conserved in **thioredoxin** sequences. The **recombinant** T. aestivum **thioredoxin** h (TrxTa) overproduced in BL21(DE3)pLysS was purified to homogeneity by a three-step procedure including heat treatment, anion-exchange chromatography and gel filtration. TrxTa showed a lower stability to high temperature than Escherichia coli **thioredoxin** or plant **thioredoxin** m. The molecular mass of TrxTa, determined by mass spectrometry, is 13,391 Da and corresponds to a protein lacking the first methionine residue, as confirmed by its N-terminal end sequence AASAAT. Using the 5,5'-dithiobis(2-nitrobenzoic acid)-reduction assay and monobromobimane revelation we showed that TrxTa is specifically reduced by wheat NADP:**thioredoxin** reductase (NTR), and not by E. coli NTR. TrxTa is able to reduce identified target proteins i.e. wheat seed alpha-amylase inhibitors (chloroform/methanol-soluble proteins). The presence of a putative transmembrane domain at the N-terminal end of the two wheat **thioredoxins** raises the question of whether these proteins are membrane anchored.

L9 ANSWER 31 OF 37 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 1999:63389 BIOSIS
DOCUMENT NUMBER: PREV199900063389
TITLE: Increased **expression** of annexin I and **thioredoxin** detected by two-dimensional gel electrophoresis of drug resistant human stomach cancer cells.
AUTHOR(S): Sinha, Pranav [Reprint author]; Huetter, Gero; Koettgen, Eckart; Dietel, Manfred; Schadendorf, Dirk; Lage, Hermann
CORPORATE SOURCE: Inst. Laboratoriumsmed. und Pathobiochem., Campus Virchow-Klinikum, Universitaetsklin. Charite, Augustenburger Platz 1, Berlin, Germany
SOURCE: Journal of Biochemical and Biophysical Methods, (Nov. 18, 1998) Vol. 37, No. 3, pp. 105-116. print.
CODEN: JBBMDG. ISSN: 0165-022X.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Feb 1999
Last Updated on STN: 16 Feb 1999

AB The therapy of advanced cancer using chemotherapy alone or in combination with radiation or hyperthermia yields an overall response rate of about 20-50%. This success is often marred by the development of resistance to cytostatic drugs. Our aim was to study the global analysis of protein **expression** in the development of chemoresistance in vitro. We therefore used a cell culture model derived from the gastric carcinoma cell line EPG 85-257P. A classical multidrug-resistant subline EPG85-257RDB selected to daunorubicin and an atypical multidrug-resistant cell variant EPG85-257RNOV selected to mitoxantrone, were analysed using two-dimensional electrophoresis in immobilized pH-gradients (pH 4.0-8.0) in the first dimension and linear polyacrylamide gels (12%) in the second dimension. After staining with coomassie brilliant blue, image analysis was performed using the PDQuest system. Spots of interest were isolated using preparative two-dimensional electrophoresis and subjected to microsequencing. A total of 241 spots from the EPG85-257RDB-standard and 289 spots from the EPG85-257RNOV-standard could be matched to the EPG85-257P-standard. Microsequencing after enzymatic hydrolysis in gel, mass spectrometric data and sequencing of the peptides after their fractionation using microbore HPLC identified that two proteins annexin I and **thioredoxin** were overexpressed in chemoresistant cell lines. Annexin I was present in both the classical and the atypical

multidrug-resistant cells. **Thioredoxin** was found to be overexpressed only in the atypical multidrug-resistant cell line.

L9 ANSWER 32 OF 37 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1997:585491 HCAPLUS
DOCUMENT NUMBER: 128:44390
TITLE: Complete genome sequence of Escherichia coli K-12
AUTHOR(S): Blattner, Frederick R.; Plunkett, Guy, III; Bloch, Craig A.; Perna, Nicole T.; Burland, Valerie; Riley, Monica; Collado-Vides, Julio; Glasner, Jeremy D.; Rode, Christopher K.; Mayhew, George F.; Gregor, Jason; Davis, Nelson Wayne; Kirkpatrick, Heather A.; Goeden, Michael A.; Rose, Debra J.; Mau, Bob; Shao, Ying
CORPORATE SOURCE: Lab. Genetics, Univ. Wisconsin-Madison, Madison, WI, 53706, USA
SOURCE: Science (Washington, D. C.) (1997), 277(5331), 1453-1462
CODEN: SCIEAS; ISSN: 0036-8075
PUBLISHER: American Association for the Advancement of Science
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The 4,639,221-base pair sequence of Escherichia coli K-12 is presented. Of 4288 protein-coding genes annotated, 38 percent have no attributed function. Comparison with five other sequenced microbes reveals ubiquitous as well as narrowly distributed gene families; many families of similar genes within E. coli are also evident. The largest family of paralogous proteins contains 80 ABC transporters. The genome as a whole is strikingly organized with respect to the local direction of replication; guanines, oligonucleotides possibly related to replication and recombination, and most genes are so oriented. The genome also contains insertion sequence (IS) elements, phage remnants, and many other patches of unusual composition indicating genome plasticity through horizontal transfer.
REFERENCE COUNT: 98 THERE ARE 98 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 33 OF 37 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1997:661238 HCAPLUS
DOCUMENT NUMBER: 127:304503
TITLE: Structures and functions of the two proteins in bovine adrenocortical mitochondria. A unique defense system against reactive oxygen species
AUTHOR(S): Hiroi, Tomoko
CORPORATE SOURCE: Radioisotope Research Institute Basic Medicine, St. Marianna University School Medicine, Kawasaki, 216, Japan
SOURCE: Sei Marianna Ika Daigaku Zasshi (1997), 25(3), 183-198
CODEN: SMIZDS; ISSN: 0387-2289
PUBLISHER: Sei-Marianna Ika Daigaku Igakkai
DOCUMENT TYPE: Journal
LANGUAGE: Japanese
AB The present author's group has reported previously that a unique protein (Mr 22,000 dalton) originally detected in the bovine adrenal cortex is a substrate protein (SP-22) for bovine ATP-dependent protease. Both SP-22 and the protease are localized in the mitochondrial matrix of bovine adrenal cortex. A third protein termed SP-22 activating protein (SAP) that activates a novel, reducing activity of SP-22 has also been found in the bovine adrenocortical mitochondria. To study the possible, biochem. interrelationship among these proteins, the present author utilized techniques of mol. biol. Firstly, oligonucleotide primers were prepared for SP-22 and SAP based on amino acid sequences fo their oligopeptides. Methods used for isolation of cDNAs were reverse transcription-polymerase chain reaction, screening of bovine adrenal medulla cDNA library, and

cloning by the rapid amplification of cDNA ends methods. Secondly, nucleotide sequences of the selected clones were determined by th dye-labeled dideoxy chain termination method in an automatic DNA sequencer. The entire nucleotide sequences determined for SP-22 and SAP were 1646 bp and 1040 bp long, resp. Amino acid sequences deduced from these nucleotide sequences revealed that both SP-22 and SAP contain mitochondrial targeting signals of 62 residues and 59 residues, resp. It was further identified that these signals are processed during their transportation into mitochondria. Both nucleotide- and amino acid sequences were compared with those of various proteins in the seven different com. databases. Protein SP-22 showed a high homol. with antioxidant protein family enzymes while SAP was completely homologous to the mammalian mitochondrial thioredoxin. Considering the chemical properties, we concluded that SP-22 is a thioredoxin-dependent peroxide reductase. Thus, the present lines of evidence indicate the presence of a new antioxidant system functioning in the mitochondria of the adrenal cortex as well as of the other tissues. In summary, this particular thioredoxin dependent system reduces hydrogen peroxide, a hazardous byproduct of the steroid-hydroxylases in the adrenal cortex as well as of Mn-superoxide dismutase in other tissues. Intramitochondrial levels of reactive oxygen species are regulated by a combined function of the ATP-dependent protease, SP-22 and SAP.

L9 ANSWER 34 OF 37 MEDLINE on STN
ACCESSION NUMBER: 97415331 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9271130
TITLE: Identification of a recombinant synaptobrevin-thioredoxin fusion protein by capillary zone electrophoresis using laser-induced fluorescence detection.
AUTHOR: Asermely K E; Broomfield C A; Nowakowski J; Courtney B C; Adler M
CORPORATE SOURCE: Pharmacology Division, U.S. Army Medical Research Institute of Chemical Defense, Aberdeen Proving Ground, MD 21010-5425, USA.
SOURCE: Journal of chromatography. B, Biomedical sciences and applications, (1997 Jul 18) Vol. 695, No. 1, pp. 67-75. Journal code: 9714109. ISSN: 1387-2273.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199709
ENTRY DATE: Entered STN: 19971008
Last Updated on STN: 19980206
Entered Medline: 19970925

AB Capillary zone electrophoresis (CZE) was utilized to identify a synaptobrevin-thioredoxin fusion protein (TSB-51). TSB-51 is a substrate for cleavage by botulinum toxin B at the Q(76)-F(77) site. TSB-51 was derivatized with a fluorophore, CBQCA [3-(4-carboxy-benzoyl)-2-quinoline-carboxaldehyde], for 4 h at room temperature. Optimal conditions for CZE separation of the TSB-51-CBQCA complex were determined: buffer (sodium borate), pH (9.0), applied voltage (25 kV), temperature (25 degrees C) and forward polarity. SDS-PAGE showed that TSB-51 had a molecular mass of approximately 19 kDa. The protein was transferred to PVDF membrane and sequenced by the Edman degradation method verifying the first twelve amino acids as SDKIIHLTDDSF. TSB-51 was also collected during CZE separation and subsequently sequenced yielding the first three amino acids as SDK. This CZE-LIF method coupled with the CBQCA derivatization, fraction collection and Edman sequencing allowed for identification of the recombinant protein, a fast separation run time and utilization of small volumes of peptide (1.5 ng protein/23.6 nl injection). This method will be used for monitoring the endopeptidase activity of botulinum toxin B on TSB-51.

L9 ANSWER 35 OF 37 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 96198118 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8626451
TITLE: Efficient **expression** of the gene for spinach phosphoribulokinase in Pichia pastoris and utilization of the **recombinant** enzyme to explore the role of regulatory cysteinyl residues by site-directed mutagenesis.
AUTHOR: Brandes H K; Hartman F C; Lu T Y; Larimer F W
CORPORATE SOURCE: Protein Engineering Program, Biology Division, Oak Ridge National Laboratory, Tennessee 37831, USA.
SOURCE: The Journal of biological chemistry, (1996 Mar 15) Vol. 271, No. 11, pp. 6490-6.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199606
ENTRY DATE: Entered STN: 19960708
Last Updated on STN: 20000303
Entered Medline: 19960624

AB Phosphoribulokinase (PRK), unique to photosynthetic organisms, is regulated in higher plants by **thioredoxin**-mediated thiol-disulfide exchange in a light-dependent manner. Prior attempts to overexpress the higher plant PRK gene in Escherichia coli for structure-function studies have been hampered by sensitivity of the **recombinant** protein to proteolysis as well as toxic effects of the protein on the host. To overcome these impediments, we have spliced the spinach PRK coding sequence immediately downstream from the AOX1 (alcohol oxidase) promoter of Pichia pastoris, displacing the chromosomal AOX1 gene. The PRK gene is now **expressed**, in response to methanol, at 4-6% of total soluble protein, without significant in vivo degradation of the **recombinant** enzyme. This **recombinant** spinach PRK is purified to homogeneity by successive anion-exchange and **dye**-affinity chromatography and is shown to be electrophoretically and kinetically indistinguishable from the authentic spinach counterpart. Site-specific replacement of all of PRK's cysteinyl residues (both individually and in combination) demonstrates a modest catalytically facilitative role for Cys-55 (one of the regulatory residues) and the lack of any catalytic role for Cys-16 (the other regulatory residue), Cys-244, or Cys-250. Mutants with seryl substitutions at position 55 display non-hyperbolic kinetics relative to the concentration of ribulose 5-phosphate. Sulfate restores hyperbolic kinetics and enhances kinase activity, presumably reflecting conformational differences between the position 55 mutants and wild-type enzyme. Catalytic competence of the C16S-C55S double mutant proves that mere loss of free sulfhydryl groups by oxidative regulation cannot account entirely for the accompanying total inactivation.

L9 ANSWER 36 OF 37 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1995:747446 SCISEARCH
THE GENUINE ARTICLE: TB584
TITLE: PURIFICATION, CHARACTERIZATION, AND CLONING OF A HEME-BINDING PROTEIN (23 KDA) IN RAT-LIVER CYTOSOL
AUTHOR: IWAHARA S (Reprint); SATOH H; SONG D X; WEBB J; BURLINGAME A L; NAGAE Y; MULLEREBERHARD U
CORPORATE SOURCE: CORNELL UNIV, COLL MED, DEPT PEDIAT, NEW YORK, NY 10021; CORNELL UNIV, COLL MED, DEPT BIOCHEM, NEW YORK, NY 10021; CORNELL UNIV, COLL MED, DEPT PHARMACOL, NEW YORK, NY 10021; ILLINOIS STATE UNIV, DEPT CHEM, NORMAL, IL 61790; UNIV CALIF SAN FRANCISCO, DEPT PHARMACEUT CHEM, SAN FRANCISCO, CA 94143; UNIV CALIF SAN FRANCISCO, CTR LIVER, SAN FRANCISCO, CA 94143; NIPPON MED COLL, DEPT INTERNAL

MED, TOKYO 113, JAPAN
COUNTRY OF AUTHOR: USA; JAPAN
SOURCE: BIOCHEMISTRY, (17 OCT 1995) Vol. 34, No. 41, pp. 13398-13406.
ISSN: 0006-2960.
PUBLISHER: AMER CHEMICAL SOC, PO BOX 57136, WASHINGTON, DC 20037-0136
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 58
ENTRY DATE: Entered STN: 1995
Last Updated on STN: 1995
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB A heme-binding protein (designated HBP23) has been purified from rat liver cytosol using heme-affinity chromatography and either reverse-phase high-performance liquid chromatography or sequential ion-exchange chromatography. The protein (23 kDa) binds heme with an affinity (K-d = 55 nM) higher than that of the abundant cytosolic heme-binding proteins, heme-binding protein (HBP)/liver fatty acid-binding protein (L-FABP) and the glutathione S-transferases (GSTs) (K-d = 100-200 nM). HBP23 is present in the cytosol of liver, kidney, spleen, small intestine, and heart, with the liver showing the highest content. A cDNA coding the 23-kDa protein was **cloned** using reverse transcription polymerase chain reaction with degenerative oligonucleotides derived from partial amino acid sequences. The **cloned** cDNA encoded 199 amino acids, and its amino acid sequence showed no homology to HBP/L-FABP, GSTs, or any other heme-binding proteins or hemeproteins. Homology search showed that HBP23 is highly homologous to mouse macrophage 23-kDa stress protein, which is inducible by oxidant stress in peritoneal macrophages [Ishii, T., Yamada, M., Sate, H., Matsue, M., Taketani, S., Nakayama, K., Sugita, Y., and Bannai, S. (1993) J. Biol. Chemical 268, 18633-18636]. **Thioredoxin** peroxidase as well as HBP23 and the mouse macrophage 23-kDa stress protein are members of the peroxiredoxin family, a recently recognized class of antioxidant proteins [Chae, H. Z., Chung, S. J., & Rhee, S. G. (1994) J. Biol. Chemical 269, 27670-27678]. An increase in HBP23 mRNA was observed in Hepa 1-6 cells after treatment with heme and cadmium and during liver regeneration after partial hepatectomy. These findings indicate an involvement of HBP23 in heme metabolism.

L9 ANSWER 37 OF 37 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1991:316120 SCISEARCH
THE GENUINE ARTICLE: FP306
TITLE: ESCHERICHIA-COLI GLUTAREDOXIN - **CLONING** AND OVEREXPRESSION, THERMODYNAMIC STABILITY OF THE OXIDIZED AND REDUCED FORMS, AND REPORT OF AN N-TERMINAL EXTENDED SPECIES
AUTHOR: SANDBERG V A (Reprint); KREN B; FUCHS J A; WOODWARD C
CORPORATE SOURCE: UNIV MINNESOTA, DEPT BIOCHEM, ST PAUL, MN 55108
COUNTRY OF AUTHOR: USA
SOURCE: BIOCHEMISTRY, (4 JUN 1991) Vol. 30, No. 22, pp. 5475-5484.
ISSN: 0006-2960.
PUBLISHER: AMER CHEMICAL SOC, 1155 16TH ST, NW, WASHINGTON, DC 20036.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 67
ENTRY DATE: Entered STN: 1994
Last Updated on STN: 1994
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Escherichia coli glutaredoxin (MW 9700) catalyzes intracellular redox reactions utilizing a disulfide/dithiol enzymatic mechanism involving the active-site residues -Cys-Pro-Tyr-Cys-. It is functionally related to the

thioredoxin family and is expected to share similar three-dimensional structure [Eklund, H., Cambillau, C., Sjoberg, B.-M., Holmgren, A., Jornvall, H., Hoog, J.-O., & Branden, C.-I. (1984) EMBO J. 3, 1443-1449]. We constructed an overexpression system in which production of glutaredoxin is controlled by temperature-sensitive **expression** of the phage T7 promoter. In addition to glutaredoxin extended by the sequence Met-Arg-Arg-Glu-Ile- at the N terminus. We have begun characterization of the structure and stability of the oxidized and reduced forms of glutaredoxin (grx-S2 and grx-(SH)2, respectively). Secondary structure calculated from CD data agrees with that predicted from the three-dimensional model of Eklund et al. The cooperative denaturation reactions of oxidized and reduced glutaredoxin were measured in temperature-induced and guanidine hydrochloride induced unfolding experiments. Surprisingly, oxidized and reduced glutaredoxins are very similar in stability. In heat-induced denaturation, monitored by CD, T(m) is 55 and 57-degrees-C for oxidized and reduced, respectively. In GuHCl denaturation, monitored by fluorescence, the midpoint denaturant concentrations are 2 M for both oxidized and reduced. It follows that the redox potentials of the disulfide bond are similar in unfolded and folded glutaredoxin. This is unexpected because in E. coli **thioredoxin** the oxidized form is far more stable than the reduced [Kelley, R. F., Shalongo, W., Jagannadham, M. V., & Stellwagen, E. (1987) Biochemistry 26, 1406-1411] and the redox potential of folded **thioredoxin** is significantly more negative than that of unfolded **thioredoxin** [Lin, T.-Y., & Kim, P. (1989) Biochemistry 28, 5282-5287].

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     (FILE 'HOME' ENTERED AT 14:18:13 ON 11 APR 2006)

     FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS,
     LIFESCI' ENTERED AT 14:18:37 ON 11 APR 2006
L1         25776 S THIOREDOXIN
L2       7634805 S CLON? OR EXPRESS? OR RECOMBINANT
L3         12217 S L1 AND L2
L4          7209 S HISTIDINE (W) TAG?
L5            83 S L3 AND L4
L6        662353 S DYE?
L7             0 S L5 AND L6
L8            48 S L3 AND L6
L9            37 DUP REM L8 (11 DUPLICATES REMOVED)

=> s inclusion (w) bod?
L10        50437 INCLUSION (W) BOD?

=> s l9 and l10
L11            1 L9 AND L10

=> d all

L11   ANSWER 1 OF 1  BIOTECHDS COPYRIGHT 2006 THE THOMSON CORP. on STN
AN    1998-09126  BIOTECHDS
TI    Production of polypeptides as inclusion bodies;
          recombinant protein preparation by plasmid pTrc99A or
          plasmid pTrxfus vector-mediated thioredoxin
          expression in Escherichia coli inclusion
          body
AU    Chatterjee D; Longo M; Flynn E; Oberfelder R
PA    Life-Technol.
LO    Rockville, MD, USA.
PI    WO 9830684 16 Jul 1998
AI    WO 1998-US492 8 Jan 1998
PRAI  US 1997-34658 8 Jan 1997
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DT Patent
LA English
OS WPI: 1998-399134 [34]
AB A new method for the preparation of a protein in the form of an **inclusion body** involves: obtaining a host cell (e.g. Escherichia coli) containing a first DNA sequence encoding the protein linked to a second DNA sequence encoding an inclusion partner (**thioredoxin** or modified **thioredoxin**), forming a gene fusion construct (plasmid pTrcpr1-monomer or pTrxA-concat); and culturing the cell to favor production of the protein as **inclusion bodies**. Also new are: vector plasmid pTrc99A and plasmid pTrxfus containing the construct; a host cell containing the vector; making a protein mol.weight ladder composition by obtaining one or more DNA sequences encoding proteins of different mol.weight values, transforming host cells with the DNA, culturing the cells to favor production of each protein, and isolating each protein; and making one or more stained proteins by incubating the proteins with one or more protein-binding **dyes** under incubation conditions to complex the proteins with the **dyes** . The methods may be used to prepare a fragment of the gene-32 protein of phage T4, a fragment of KpnI-methylase or a fragment of E. coli Dead-Box protein or **thioredoxin**. (84pp)
CC D PHARMACEUTICALS; D3 Peptides and Proteins; A GENETIC ENGINEERING AND FERMENTATION; A1 Nucleic Acid Technology; L PURIFICATION; L1 Downstream Processing
CT PHAGE T4 GENE-32 PROTEIN, KPNI DNA-METHYLASE, ESCHERICHIA COLI DEAD-BOX PROTEIN, **THIOREDOXIN RECOMBINANT** FUSION PROTEIN PREP., PROTEIN PURIFICATION, VECTOR PLASMID PTRCPR1-MONOMER, PLASMID PTRXA-CONCAT **EXPRESSION** IN ESCHERICHIA COLI **INCLUSION BODY**, APPL. MOL.WT. LADDER CONSTRUCTION, PROTEIN **DYEING** ENZYME DNA SEQUENCE PROTEIN SEQUENCE BACTERIUM **CLONING** (VOL.17, NO.21)

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(FILE 'HOME' ENTERED AT 14:18:13 ON 11 APR 2006)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS, LIFESCI' ENTERED AT 14:18:37 ON 11 APR 2006

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L1        25776 S THIOREDOXIN
L2      7634805 S CLON? OR EXPRESS? OR RECOMBINANT
L3        12217 S L1 AND L2
L4         7209 S HISTIDINE (W) TAG?
L5           83 S L3 AND L4
L6       662353 S DYE?
L7            0 S L5 AND L6
L8           48 S L3 AND L6
L9           37 DUP REM L8 (11 DUPLICATES REMOVED)
L10       50437 S INCLUSION (W) BOD?
L11           1 S L9 AND L10
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=> e chatterjee d k/au

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E2           36     CHATTERJEE D J/AU
E3          381 --> CHATTERJEE D K/AU
E4           54     CHATTERJEE D N/AU
E5           23     CHATTERJEE D P/AU
E6            3     CHATTERJEE D R/AU
E7           55     CHATTERJEE D S/AU
E8            7     CHATTERJEE DANIEL/AU
E9            5     CHATTERJEE DAS J M/AU
E10          16     CHATTERJEE DAS S/AU
E11           1     CHATTERJEE DAS SASWATI/AU
E12           5     CHATTERJEE DEB/AU
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               EE DANIEL"/AU OR "CHATTERJEE DAS J M"/AU OR "CHATTERJEE DAS S"/AU
                OR "CHATTERJEE DAS SASWATI"/AU OR "CHATTERJEE DEB"/AU)

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E3          698 --> LONGO M/AU
E4          113      LONGO M A/AU
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E6           23      LONGO M C/AU
E7            2      LONGO M CASTELLANI/AU
E8           14      LONGO M D/AU
E9            2      LONGO M D C/AU
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E11          20      LONGO M G/AU
E12          15      LONGO M I/AU

=> s e3
L13         698 "LONGO M"/AU

=> e oberfelder r/au
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E2            1      OBERFELDARZT/AU
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E4           30      OBERFELDER R W/AU
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E6            5      OBERFELDER ROBERT W/AU
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E8           15      OBERFELL C R/AU
E9           10      OBERFELL CHARLES R/AU
E10           1      OBERFELL CHAS R/AU
E11          73      OBERFELL G G/AU
E12           1      OBERFELL G S/AU

=> s e3-e7
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                /AU OR "OBERFELDER ROBERT W"/AU OR "OBERFELDER ROBERT WILLIAM"/A
               U)

=> e flynn e/au
E1            1      FLYNN DUANE J/AU
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E4           54      FLYNN E A/AU
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E6            5      FLYNN E C/AU
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E8            3      FLYNN E E/AU
E9            1      FLYNN E F/AU
E10          11      FLYNN E H/AU
E11         151      FLYNN E J/AU
E12           1      FLYNN E J */AU

=> s e3
L15         285 "FLYNN E"/AU

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     FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS,
     LIFESCI' ENTERED AT 14:18:37 ON 11 APR 2006
L1         25776 S THIOREDOXIN
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L7             0 S L5 AND L6
L8            48 S L3 AND L6
L9            37 DUP REM L8 (11 DUPLICATES REMOVED)
L10        50437 S INCLUSION (W) BOD?
L11            1 S L9 AND L10
                 E CHATTERJEE D K/AU
L12          550 S E3-E12
                 E LONGO M/AU
L13          698 S E3
                 E OBERFELDER R/AU
L14           56 S E3-E7
                 E FLYNN E/AU
L15          285 S E3

=> s l11 or l12 or l13 or l14 or l15
L16        1578 L11 OR L12 OR L13 OR L14 OR L15

=> s l9 and l16
L17            1 L9 AND L16

=> d ibib ab

L17   ANSWER 1 OF 1  BIOTECHDS COPYRIGHT 2006 THE THOMSON CORP. on STN
ACCESSION NUMBER: 1998-09126  BIOTECHDS
TITLE:            Production of polypeptides as inclusion
                  bodies;
                       recombinant protein preparation by plasmid
                     pTrc99A or plasmid pTrxfus vector-mediated
                     thioredoxin expression in Escherichia
                     coli inclusion body
AUTHOR:           Chatterjee D; Longo M; Flynn E;
                  Oberfelder R
PATENT ASSIGNEE:  Life-Technol.
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PRIORITY INFO:    US 1997-34658 8 Jan 1997
DOCUMENT TYPE:    Patent
LANGUAGE:         English
OTHER SOURCE:     WPI: 1998-399134 [34]
AB    A new method for the preparation of a protein in the form of an
      inclusion body involves: obtaining a host cell (e.g.
      Escherichia coli) containing a first DNA sequence encoding the protein
      linked to a second DNA sequence encoding an inclusion partner (
      thioredoxin or modified thioredoxin), forming a gene
      fusion construct (plasmid pTrcpr1-monomer or pTrxA-concat); and culturing
      the cell to favor production of the protein as inclusion
      bodies.  Also new are: vector plasmid pTrc99A and plasmid pTrxfus
      containing the construct; a host cell containing the vector; making a
      protein mol.weight ladder composition by obtaining one or more DNA sequences
      encoding proteins of different mol.weight values, transforming host cells
      with the DNA, culturing the cells to favor production of each protein,
      and isolating each protein; and making one or more stained proteins by
      incubating the proteins with one or more protein-binding dyes
      under incubation conditions to complex the proteins with the dyes
      .  The methods may be used to prepare a fragment of the gene-32 protein
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of phage T4, a fragment of KpnI-methylase or a fragment of E. coli Dead-Box protein or **thioredoxin**. (84pp)

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     (FILE 'HOME' ENTERED AT 14:18:13 ON 11 APR 2006)

     FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS,
     LIFESCI' ENTERED AT 14:18:37 ON 11 APR 2006
L1         25776 S THIOREDOXIN
L2       7634805 S CLON? OR EXPRESS? OR RECOMBINANT
L3         12217 S L1 AND L2
L4          7209 S HISTIDINE (W) TAG?
L5            83 S L3 AND L4
L6        662353 S DYE?
L7             0 S L5 AND L6
L8            48 S L3 AND L6
L9            37 DUP REM L8 (11 DUPLICATES REMOVED)
L10        50437 S INCLUSION (W) BOD?
L11            1 S L9 AND L10
                 E CHATTERJEE D K/AU
L12          550 S E3-E12
                 E LONGO M/AU
L13          698 S E3
                 E OBERFELDER R/AU
L14           56 S E3-E7
                 E FLYNN E/AU
L15          285 S E3
L16         1578 S L11 OR L12 OR L13 OR L14 OR L15
L17            1 S L9 AND L16
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	Issue Date	Pages	Document ID	Title
1	20060406	70	US 20060073530 A1	Methods and compositions involving sortase B
2	20060216	83	US 20060035315 A1	Transporters and ion channels
3	20051020	22	US 20050233357 A1	Linking gene sequence to gene function by three dimensional (3D) protein structure determination
4	20050526	32	US 20050112637 A1	Mutant DNA polymerases and uses thereof
5	20050127	39	US 20050019829 A1	Protein/solubility folding assessed by structural complementation
6	20041216	117	US 20040253701 A1	Protein and peptide fragments from mouse telomerase reverse transcriptase
7	20041209	144	US 20040248251 A1	Receptors and membrane associated proteins
8	20041111	171	US 20040224911 A1	Transporters and ion channels
9	20041014	31	US 20040204563 A1	Methods for production of proteins
10	N/A, Contact help desk	31	US 20040157291 A1	
11	N/A, Contact help desk	72	US 20040152874 A1	
12	N/A, Contact help desk	46	US 20040127683 A1	

13	20040617	140	US 20040116666 A1	Transporters and ion channels

	Issue Date	Pages	Document ID	Title
14	20040318	182	US 20040053258 A1	Transporters and ion channels
15	20040226	87	US 20040037846 A1	Chlamydia pmp proteins, gene sequences and uses thereof
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17	20040205	73	US 20040023207 A1	Assays for drug discovery based on microcompetition with a foreign polynucleotide
18	20040205	72	US 20040023206 A1	Methods for chronic disease diagnosis based on microcompetition with a foreign polynucleotide
19	20040205	63	US 20040022764 A1	Inhibition of microcompetition with a foreign polynucleotide as treatment of chronic disease
20	20040122	119	US 20040014945 A1	Transporters and ion channels
21	20040101	40	US 20040002065 A1	PROTEIN/SOLUBILITY FOLDING ASSESSED BY STRUCTURAL COMPLEMENTATION
22	20031120	144	US 20030216310 A1	Transporters and ion channels
23	20031120	43	US 20030215915 A1	Cytochrome P450 expression in enterobacteria
24	20031113	94	US 20030211499 A1	Transporters and ion channels

25	20031023	96	US 20030198944 A1	Compositions and methods for reverse transcription of nucleic acid molecules

	Issue Date	Pages	Document ID	Title
26	20031002	96	US 20030186270 A1	Compositions and methods for reverse transcription of nucleic acid molecules
27	20030911	121	US 20030171275 A1	Transporters and ion channels
28	20030911	31	US 20030170622 A1	GENERATION OF SPECIFIC BINDING PARTNERS BINDING TO (POLY)PEPTIDES ENCODED BY GENOMIC DNA FRAGMENTS OR ESTS
29	20030213	97	US 20030032086 A1	COMPOSITIONS AND METHODS FOR REVERSE TRANSCRIPTION OF NUCLEIC ACID MOLECULES
30	20021107	37	US 20020164736 A1	Ginkgo biloba levopimaradiene synthase
31	20020627	97	US 20020081581 A1	COMPOSITIONS AND METHODS FOR REVERSE TRANSCRIPTION OF NUCLEIC ACID MOLECULES
32	20020530	35	US 20020065392 A1	METHODS FOR PRODUCTION OF PROTEIN
33	20010823	22	US 20010016314 A1	LINKING GENE SEQUENCE TO GENE FUNCTION BY THREE DIMESIONAL (3D) PROTEIN STRUCTURE DETERMINATION
34	20060124	92	US 6989259 B2	Recombinant methods for making reverse transcriptases and mutants thereof
35	20050920	80	US 6946283 B2	Ginkgo biloba levopimaradiene synthase

36	20041228	90	US 6835561 B1	Composition of reverse transcriptases and mutants thereof

	Issue Date	Pages	Document ID	Title
37	20040727	112	US 6767719 B1	Mouse telomerase reverse transcriptase
38	20040427	36	US 6727070 B2	Protein/solubility folding assessed by structural complementation
39	20040309	29	US 6703484 B2	Methods for production of proteins
40	20031125	30	US 6653068 B2	Generation of specific binding partners binding to (poly)peptides encoded by genomic DNA fragments or ESTs
41	20030520	42	US 6566108 B1	Expression of functional cytochrome P450 monooxygenase system in enterobacteria
42	20030211	96	US 6518019 B2	Compositions and methods for reverse transcription of nucleic acid molecules
43	19991012	19	US 5965399 A	Cloning and expression of rat liver and porcine liver ribonuclease inhibitor
44	19990803	34	US 5932440 A	Mammalian ribonuclease inhibitors and use thereof
45	19990720	28	US 5925523 A	Intraction trap assay, reagents and uses thereof
46	19931214	26	US 5270179 A	Cloning and expression of T5 DNA polymerase reduced in 3'- to-5' exonuclease activity

	L #	Hits	Search Text
1	L1	1	"5270181".pn.
2	L2	10	inclusion adj partner
3	L3	0	l1 and l2
4	L4	278274	dye$2
5	L5	0	l1 and l4
6	L6	5827	thioredoxin
7	L7	830687	clon$3 or express$3 or recombinant
8	L8	4203	l6 same l7
9	L9	15	l4 same l8
10	L10	333343	CHATTERJEE LONGO FLYNN OBERFELDER
11	L11	210	l6 and l10
12	L12	7526	inclusion adj bod$3
13	L13	46	l11 and l12